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Tyson's Site
Montgomery County, Pennsylvania

Off-Site Operable Unit Remedial Investigation Report

Volume V
Endangerment Assessment

July 29, 1987

AR301364

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SECTION 1

INTRODUCTION

1.1 Purpose of CERCLA Endangerment Assessments

The need to include estimates of risk in the decision-making process for contaminated sites has been recognized by the U.S. Environmental Protection Agency (EPA), and is now a required part of the Comprehensive Environmental Resource Conservation and Liability Act (CERCLA) RI/FS process. Endangerment assessment evaluates the demographic, geographic, physical, chemical, and biological factors at a site to determine whether there is a risk to public health or the environment.

The process can be used to evaluate the current risk as well as the risk that might be associated with future actions, such as potential remedial measures. Thus quantitatively derived estimates of risk may be used to determine if present conditions pose a health/environmental threat, and what effect on that risk various remedial actions might have.

1.2 Objectives

The Tyson's Site has been subdivided by EPA into On-Site and Off-Site areas, with the Off-Site Area comprising several separate Operable Units. The locations of the Operable Units are presented in Figures 1-1 and 1-2.

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Figure 1-1
Off-Site Operable Units
Tyson's Site

LEGEND

—————— Tyson's Site Boundary

············· Approximate Location of
Leachate Collection System
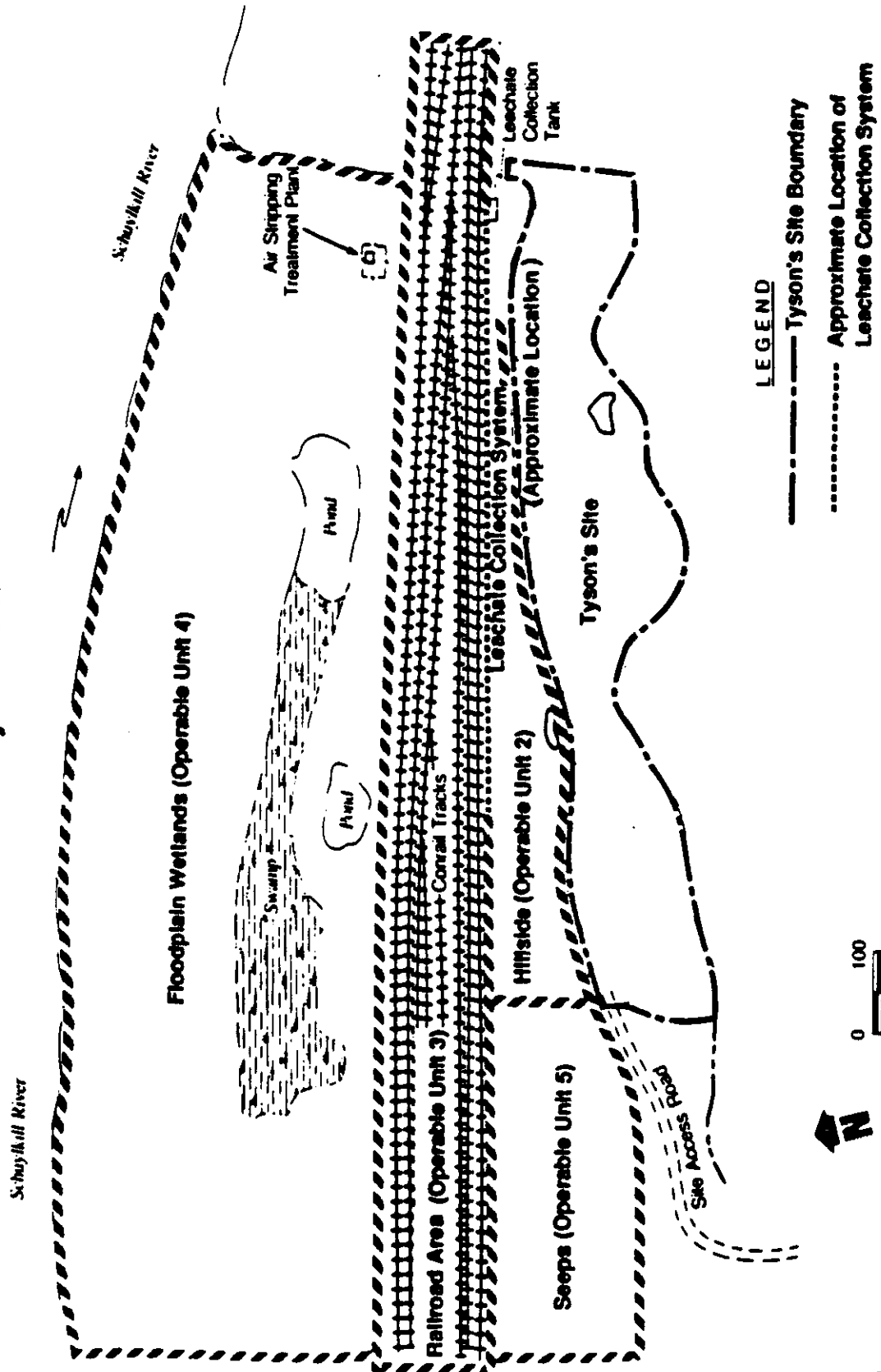
Figure 1-2
Generalized Cross Section of Operable Off-Site Units
Tyson's Site

LEGEND

-- -- Top Of Bedrock

☐ Clayey Sand To Sandy Silt With Some Gravel

▨ Fine To Medium Sand With Some Gravel, Slightly Silty

Vertical Exaggeration: 2x
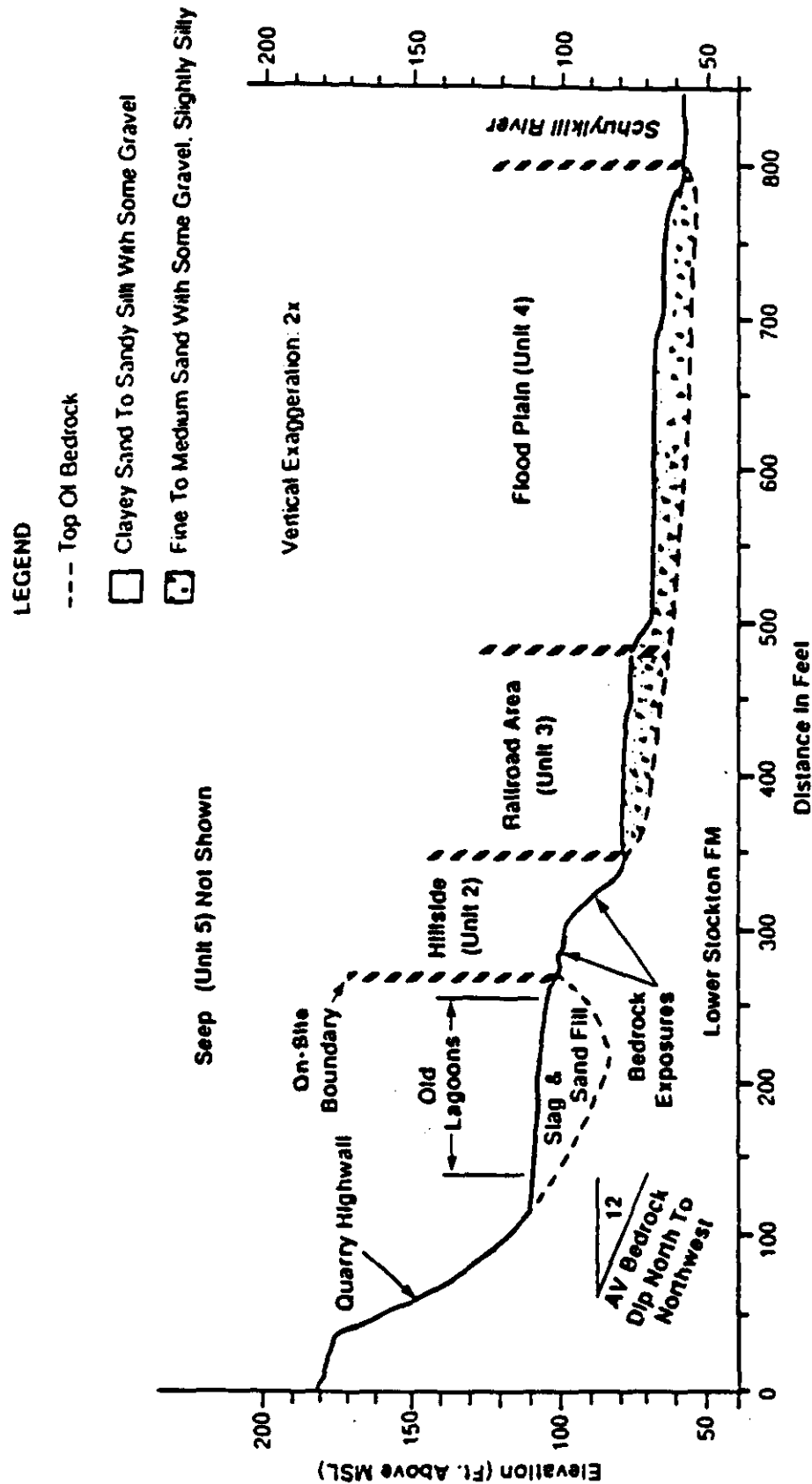
This Endangerment Assessment (EA) evaluates the level of risk to
human populations and the environment from the following Off-Site
Operable Units:

- the Deep Aquifer (Operable Unit 1),

- the Hillside Area (Operable Unit 2),

- the Railroad Area (Operable Unit 3),

- Floodplain/Wetlands Area (Operable Unit 4), and

- the Seep Area (Operable Unit 5).

This assessment considers risks from potential carcinogens and
noncarcinogens and compares concentrations of contaminants under
current conditions with applicable and relevant environmental
standards. The existing risks evaluated in this assessment will
be considered in a subsequent Off-Site Operable Unit Feasibility
Study (FS), which will discuss the degree and type of remediation
required for each of the Off-Site Operable Units.

1.3 Site Background

1.3.1 Site Description

Tyson's Site is an abandoned septic and chemical waste disposal
site reported to have operated from 1962 to 1970 within a

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sandstone quarry. The site is located in Upper Merion Township, Montgomery County, Pennsylvania (Figure 1-3). The approximate four-acre site which constitutes a series of former unlined lagoons, is bordered on the east and west by unnamed tributaries to the Schuylkill River, a steep quarry high-wall to the south, and a Conrail railroad switching yard to the north. North of the Conrail tracks is the Schuylkill River floodplain. The area of the former lagoons lies above the 100-year floodplain.

1.3.2 History of Site Operations

From 1960 to 1970, the site was owned and operated by companies owned by Franklin P. Tyson and Fast Pollution Treatment, Inc. The stock of this corporation was owned by the current owner of the land, General Devices, Inc. (GDI), and by Franklin P. Tyson. GDI was active in the management of Fast Pollution Treatment, Inc. The site was used for disposal of liquid septic tank waste and sludges and chemical wastes which were hauled to the site in bulk tank trucks. It appears that as the lagoons were filled with wastes and subsequently covered, new lagoons were created. Figure 1-2 shows the locations of the former lagoons as interpreted from 1965 and 1973 aerial photographs of the area. In 1969, the property was purchased from Fast Pollution Treatment, Inc. by GDI. The Pennsylvania Department of Environmental Resources (PA DER) ordered the site owners, GDI, to close the facility in 1973. During closure, the lagoons were reported to be emptied of standing water, backfilled, vegetated, and the contents transported off site.

Figure 1-3
Location Map
Tyson's Site

Norristown

Schuylkill River

Barbadoest

READING

BROWNIE

Site

Quad Source: U.S.G.S Norristown, PA

0   1000   2000
Scale In Feet

N.E. Extension
Penn. Tumpike

Pottstown

100

Penn. Turnpike

Norristown

King Of Prussia

SITE

Delaware River

1

202

100

30

30

West Chester

202   1

PHILADELPHIA

PENNSYLVANIA

QUADRANGLE LOCATION

0   5   10

Scale In Miles

N

ERM
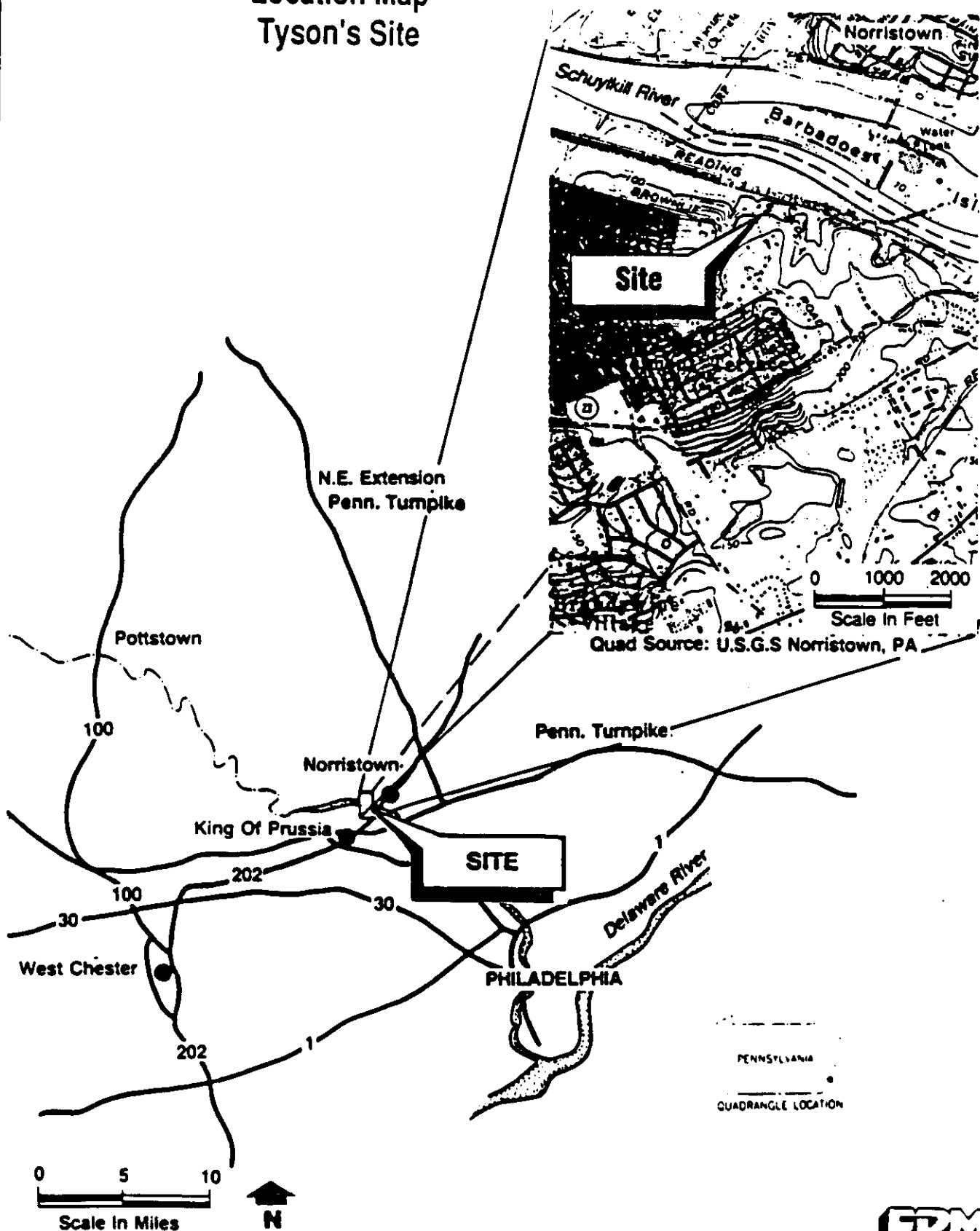
1.3.3 Site Geology

Three types of geologic materials were encountered during the investigation: the overburden materials south of the railroad tracks, the floodplain deposits north of the railroad tracks, and the Lower Member of the Stockton Formation which underlies all of the unconsolidated materials within the area of the investigation.

1.3.3.1 Overburden Materials South of the Railroad Tracks

Overall, the topography of the area south of the railroad tracks can be described as two terraces with two intervening step slope sections. The lower terrace, closest to the tracks,has been disturbed by previous site activities. Although the Off-Site Operable Unit RI focused primarily on the areas north of the former lagoons, an understanding of the relationship of the overburden materials in the area of the lagoons to the underlying bedrock aquifer and other off-site areas is critical to an overall understanding of the pathways for contaminant migration. Information presented in this section is derived from the previous investigations and the accompanying RI. The results of the previous investigations are reported in the following documents:

- "Remedial Investigation Report and Feasibility Study Work Plan for Tyson's Dump Site, Montgomery County, Pennsylvania", Baker/TSA, August 1984.

- "Supplemental Site Assessment Tyson's Dump Superfund Site, King of Prussia, Pennsylvania", conducted by Woodward-Clyde Consultants (August, 1985), and

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-    SRW Associates, Inc. Report of Additional Subsurface
     Exploration and Analysis, (November, 1985).

The overburden materials south of the railroad tracks can be
divided into three types of materials: undisturbed colluvial
deposits, fill material implaced during past remedial activities
at the former lagoon areas, and construction debris and fill
material in the seep area. Figure 1-4 shows the approximate
distribution of the various overburden materials.

The undisturbed overburden deposits generally consist of a thin
topsoil overlying the colluvial deposits and weathered bedrock.
The topsoil is an organic rich silty sand. The colluvial
materials and weathered bedrock are comprised of sandy silts with
some clays. Some fine to coarse gravel is also found at depth in
the inconsolidated deposits. The thickness of the colluvial
material varies greatly over the area, from thrity-one and
one-half feet at the eastern border of the site to absent, where
bedrock outcrops between the eastern and western sets of lagoons.

The overburden materials within the former lagoon area are
primarily fill materials of silty, gravelly sand, quarry rubble,
possible residual sludges, construction debris and colluvium.
The materials were emplaced during the past disposal and remedial
activities at the site. Topsoil in these areas is thin and often
discontinuous. The former lagoon area can best be described as
two bowl-like depressions in the bedrock surface separated by a
bedrock high. The western set of lagoons is divided into two
depressions separated by a second high bedrock. The thickness of
the fill material within these depressions varies from a maximum
of twenty-five feet to absent at the bedrock highs. The bedrock
highs are actual bedrock outcrops.

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**Figure 1-4
Distribution of Overburden Deposits
South of Railroad Tracks
Tyson's Site**
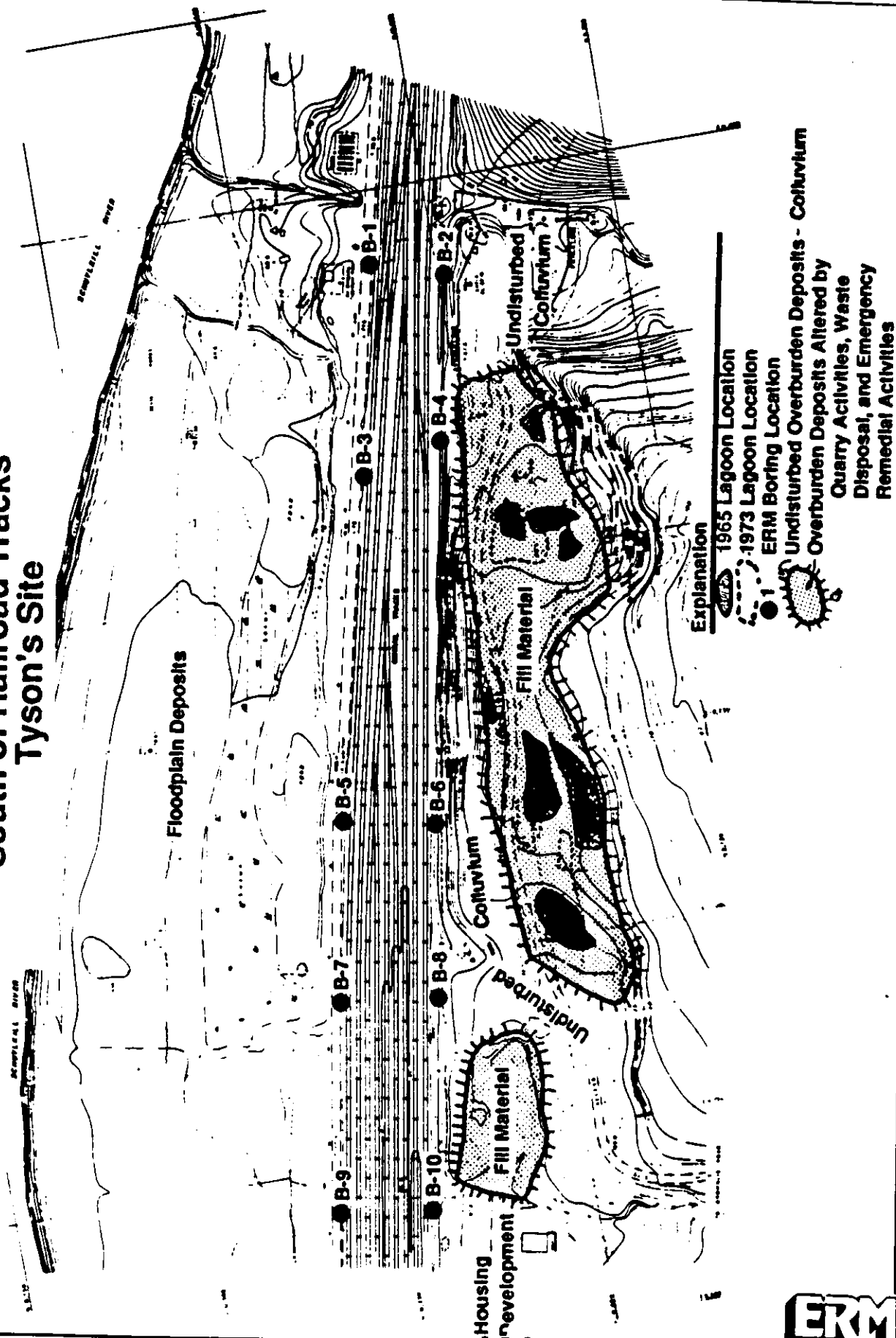
The overburden materials encountered during installation of the
test pits in the seep area included a mixture of disturbed and
undisturbed colluvial deposits and construction debris. The
construction debris was comprised of cinder blocks, wood, glass,
and plastic materials. The greatest thickness of fill material
in the seep area is about six feet. The undisturbed colluvial
deposits discussed above were encountered below the fill material
to about eleven feet (the maximum depth of the test pits).
Bedrock was not encountered in any of the test pits.

1.3.3.2 Floodplain Deposits

The Schuylkill River floodplain begins at the base of the bedrock
outcrop just north of the former lagoon area, essentially
parallel to and immediately south of the Conrail tracks (Figure
1-4). With the exception of the ravine to the far east of the
lagoons, the thickness of the floodplain deposits beneath the
railroad tracks varies from three to ten feet. The thickness of
these deposits at Boring B-2 (Figure 1-4) near the eastern edge
of the site was 31.5 feet, nearly three times that at any other
boring completed south of the Conrail tracks. The greater depth
to bedrock at this location corresponds with similar results
obtained in past investigations by Woodward-Clyde Consultants.
This appears to be the result of a zone of increased bedrock
weathering which has resulted from concentrated fracturing in the
bedrock. The large ravine adjacent to these two borings is
probably a result of the enhanced weathering.

As shown on Figure 1-5, depth to bedrock beneath the railroad
tracks drops sharply from three to ten feet at the base of the
embankment south of the railroad tracks to greater than twenty
feet on the north side of the railroad tracks. From the north

Figure 1-5
North-South Cross Section
Unconsolidated Deposits
Tyson's Site
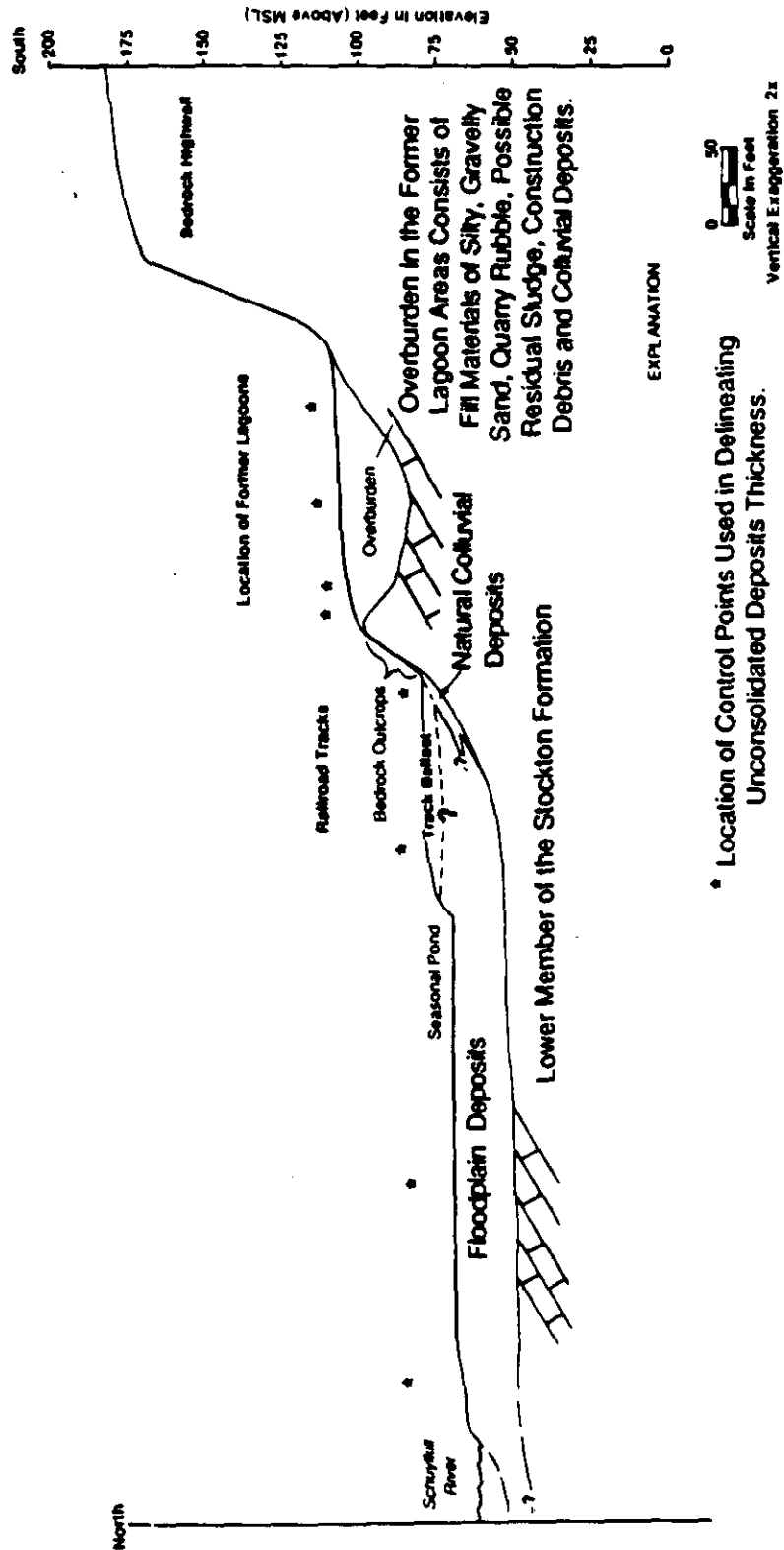
side of the railroad tracks to the edge of the Schuylkill River, the thickness of these deposits remains relatively constant and is typically in the range of twenty to twenty-five feet.

The railroad tracks are supported by a ballast which ranges between 1.5 and 9.4 feet in thickness. The ballast consists of very coarse crushed stone (limestone) with a dark very fine cinder matrix. Floodplain deposits underlying the ballast are comprised of interbedded silty, sandy clay, white coarse gravel, and gravel-sized clasts of weathered arkosic sandstone. It appears that materials in this area actually represent a transition between the colluvial deposits originating from the steep hillside and the floodplain deposits. The floodplain deposits north of the railroad tracks can be divided into three sub-units as follows:

- The upper one to two feet of organic rich silty clay;

- Ten to fifteen feet of brownish red sandy clays, sometimes mottled with some silt and trace gravel and cobbles (this material tends to become coarser toward the river with some boulders); and

- A basal sand and gravel unit with some cobbles which extends to the top of bedrock. This unit may be as much as ten feet thick at the river, but is absent at the railroad tracks.

1.3.4 Demography

Upper Merion Township has a population of approximately 26,000. This represents an increase of 308 percent since 1950, with an average annual increase of 10 percent since 1970. The population of the Township from 1940 to 1980 is presented below (Supervisors of Upper Merion Township, 1985):

Census Year	Population
1940	6,143
1950	6,404
1960	17,096
1970	23,743
1980	26,138

Much of the growth in this Township has been attributed to expansion of commercial enterprises from the city of Philadelphia, as well as movement of people from Philadelphia or other large east coast cities, to this small (16.8 sq. miles) community.

The population of the Township is comprised primarily of young, middle-income families working in the Philadelphia or King of Prussia areas. Only 19 percent of the Township is age 55 or older.

In 1980, the work force within Upper Merion consisted of 33,000 people; it is estimated that by the year 2000, 47,294 people will be employed within township boundaries. Employment is primarily at the professional/managerial level, in the commercial or office sectors.

1.3.5 Land Use

There are approximately 10,500 acres of land in Upper Merion Township. Of this, 31 percent (3,254 acres) is devoted to residential use and 22 percent is under either commercial/industrial or institutional/recreational development. Commercial/industrial land uses include shopping centers, office complexes, light industry, and quarry operations. Institutional/recreational development includes churches, schools, cemeteries, municipal facilities, as well as parks and open space. Eighteen percent of the Township's total land area is considered open space.

The site is located in the Belmont Planning Area of the Township and is presently inactive; however, a subdivision with 58 single family homes has recently been constructed to the west and adjacent to the site. The majority of land within the 840 acre Belmont area is similarly devoted to single family, detached housing units at a density of 2.87 dwellings per acre. The development of vacant parcels in this planning area is limited due to terrain features (steep slopes) or flooding potential.

Other residential and commercial areas are located within close proximity of the site. These include Norristown, Pennsylvania, located across the Schuylkill River approximately one-half mile northeast of the site, and Bridgeport, Pennsylvania, one mile east of the site on the Schuylkill River.

1.3.6 Natural Resources

The area around the Tyson's Site supports a diverse flora and fauna. Vegetation observations made during a site reconnaissance are presented in the RI report. Vegetation types range from

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upland species along the railroad and higher elevations to floodplain/wetland assemblages in the lower elevations where the plants range from obligate wetland species to faculative upland species.

During the reconnaissance survey twenty-six (26) species of birds were also observed. During the course of additional field work several waterfowl were observed in the pond associated with the wetland area. A pair of Canada geese and Mallard ducks were observed nesting in the pond/adjacent wetland. Ring-necked pheasants (both juvenile and adult) were commonly sighted. Pickeral frogs and green frogs were observed in the pond/wetland and in ponded areas on most drainage ditches as well as along the shore of the Schuylkill River. Snapping turtles were captured for tissue analysis from the Schuylkill River.

Signs or actual sightings of mammals included opossum, cottontail rabbit, gray squirrel, skunk, racoon, muskrat, and white-tailed deer.

Harvest of terrestrial resources by hunting is restricted by township ordinance forbidding the discharge of firearms. Mr. William Wasserman (Pennsylvania Game Commission - Game Protector) in a telephone conversation on April 24, 1987, indicated some illegal hunting still takes place despite the restrictions. No survey or estimate of hunter success was available. Mr. Wasserman suggested that the trapping of muskrats for fur is possible in the area. He had no idea if the meat was consumed.

Based on Mr. Wasserman's knowledge of the area, habitat quality, and reduced hunting pressure, Mr. Wasserman would characterize the wildlife resources in the area as good.

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The Schuylkill River supports a number of game fish in the site vicinity. Telephone conversation with Mr. Mike Kaufmann (Pennsylvania Fish Commission - Fisheries Manager - Quakertown) indicated that the following game fish are actively sought in the area: muskellunge, large-mouth bass, channel catfish, bullhead catfish, rock bass, and bluegills. In addition, carp are harvested for consumption as well as Corbicula clams by certain groups.

Due to the presence of two public boat launch areas (Norristown and Valley Forge Park) the area receives considerable fishing pressure from boaters as well as from shore fishermen. The large mouth bass population is good enough to support bass tournaments. Mr. Kaufmann was unaware of any creel census or fishing effort studies in the area, but characterized fishing pressure as high.

Historically, the Schuylkill River supported an anadromous fisheries. The construction of dams below the site stopped any anadromous fish from migrating. Future efforts to breach the dams or equip them with fish ladders could restore the anadromous fish populations. In anticipation of the potential return of unrestricted access to and from saline waters, the Pennsylvania Fish Commission had, in 1986, stocked fingerling shad at several locations in the river upstream of the site for home stream imprinting. Schuylkill imprinted juveniles are not expected to return from the sea for 4-6 years. According to Mr. Kaufmann, the permitting process and local opposition may delay the fish ladders planned for the Flatrock Dam and the planned breaching of the Plymouth Dam. The Norristown Dam may be eventually equipped with fish ladders as part of the development as a hydropower facility.

No threatened or endangered species of birds and mammals or fishes, amphibians and reptiles are known to occur on or in the vicinity of the site. This determination is based on letters of inquiry to Mr. Jacob Sitlinger (Pennsylvania Game Commission - Birds and Mammals) and Mr. Clark Shiffer (Pennsylvania Fish Commission - Fish, Amphibians and Reptiles).

Information provided by the Pennsylvania National Diversity Inventory (Ms. Kathleen Regan) indicated that no threatened plants are known to occur on or in the vicinity of the site.

The pool created by the Norristown Dam along with two public boat launch areas has resulted in heavy boating usage both for fishing and recreation. The deeper areas are popular for water skiing and pleasure cruising. Children have been observed wading, generally in the boat launch areas.

1.3.7 Climatology

The climate of Montgomery County is characterized by warm, humid summers, moderately cold winters, and ample rainfall. The average annual temperature ranges from 32°F in January to 77°F in July (Smith and Soil Survey Staff, 1967). Average minimum and maximum temperatures during the period of 1951 to 1980, as recorded at the Phoenixville Station (the closest temperature recording NOAA station, located approximately 15 miles northwest of the site), are presented in Table 1-1.

The average annual precipitation for Montgomery County, including both rainfall and the water equivalent of melting snow, is 42 inches. Precipitation normals during the period of 1951-1980, as

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TABLE 1-1

Historical Temperature and Precipitation

Temperature ($^{\circ}$F) as recorded at Phoenixville, PA
(1951-1980)

Month	Average Daily Maximum	Average Daily Minimum	Mean
January	40.0	20.1	30.1
February	42.9	21.6	32.2
March	52.5	30.1	41.3
April	65.1	39.1	52.1
May	75.3	48.9	62.1
June	83.2	57.8	70.5
July	87.4	62.3	74.9
August	85.8	60.6	73.2
September	78.9	53.9	66.5
October	67.7	42.3	55.0
November	55.4	33.7	44.6
December	43.9	24.7	34.3
Annual	64.8	41.3	53.1

Source: NOAA, 1980

Average precipitation (inches) as recorded at Norristown, PA
(1951-1980)

Month	Precipitation
January	3.29
February	2.95
March	4.07
April	3.63
May	3.64
June	3.59
July	4.18
August	4.46
September	4.10
October	3.18
November	3.65
December	3.71
Annual	44.45

Source: NOAA, 1980

recorded at the Norristown Station (the closest precipitation recording NOAA station, located approximately one-half mile northeast of the site), are presented in Table 1-1.

Variations in temperature and precipitation across the county do occur. For example, Phoenixville had an average annual precipitation of 43.55 inches between 1951 and 1980, whereas Norristown averaged 44.45 inches during the same period. These variations, similar to those which occur in temperature, are attributed to differences in local relief. The range in elevation in Montgomery County is 100-400 feet; minimum temperature readings tend to be lower in valleys, whereas precipitation is somewhat lower in areas of higher elevation. Weather patterns are also occasionally influenced by the Atlantic Ocean, which is approximately 75 miles southeast of the site (Smith and Soil Survey Staff, 1967).

1.3.8 Flood Potential

Flood elevations (Flood Insurance Study, Township of Upper Merion) for the Schuylkill River, the major waterway in the vicinity of the site, as recorded approximately one mile upstream from the site at the Norristown Dam, are:

Flood Frequency	Elevation (feet above MSL)
10 year	70.5
50 year	77
100 year	80
500 year	87

The site, located approximately 110 feet above mean sea level (MSL), is not located within the floodplain; but the railroad (80

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feet above MSL) and other areas downgradient from the site are located within the 100 year floodplain.

The average discharge of the Schuylkill River, as recorded at Pottstown, Pennsylvania (the nearest U.S. Geological Survey gauging station, approximately twenty-five miles upstream from the site) for a 57 year period (1926-1983) was 1,890 cubic feet per second (cfs). The maximum and minimum discharges recorded at Pottstown during this period were 95,900 cfs and 175 cfs, respectively (White et. al., 1984).

1.3.9 Site Drainage

The site is located in an abandoned sandstone quarry, approximately 550 feet south of the Schuylkill River. The sandstone quarry was excavated into the side of a small ridge. This old excavation takes the form of a bench, thirty to sixty feet above the Schuylkill River. Directly below the quarry is a railroad switching yard and the Schuylkill River floodplain.

The areas formerly occupied by the lagoons are not affected by natural drainage channels. Minor regrading of the former lagoon areas by the EPA enhanced surface drainage and prevented water from ponding in the former lagoon area, minimizing infiltration into the soils. A leachate collection system installed by EPA to collect much of the surface water drainage originating from the site. This work was completed as part of the immediate removal actions in March, 1983.

Two unnamed tributaries to the Schuylkill River are located to the east and west of the former lagoon areas (Figure 1-6). The eastern tributary occupies a large north-south trending ravine

Figure 1-6
Drainage Features
Tyson's Site

Barbadoes Island

Schuyikill River

Pond

Pond

Air Stripper

Conrail Tracks (Multiple)

Housing Development

Spring

100

Former Lagoons

Western Tributary

Eastern Tributary

150

100

Brownie

Swim Club

150

Road

200

200

Anderson Road

Scale in Feet

0   250   500

N

EXPLANATION

Fenced Area

Inferred Drainage

Well-Defined Drainage

Swamp

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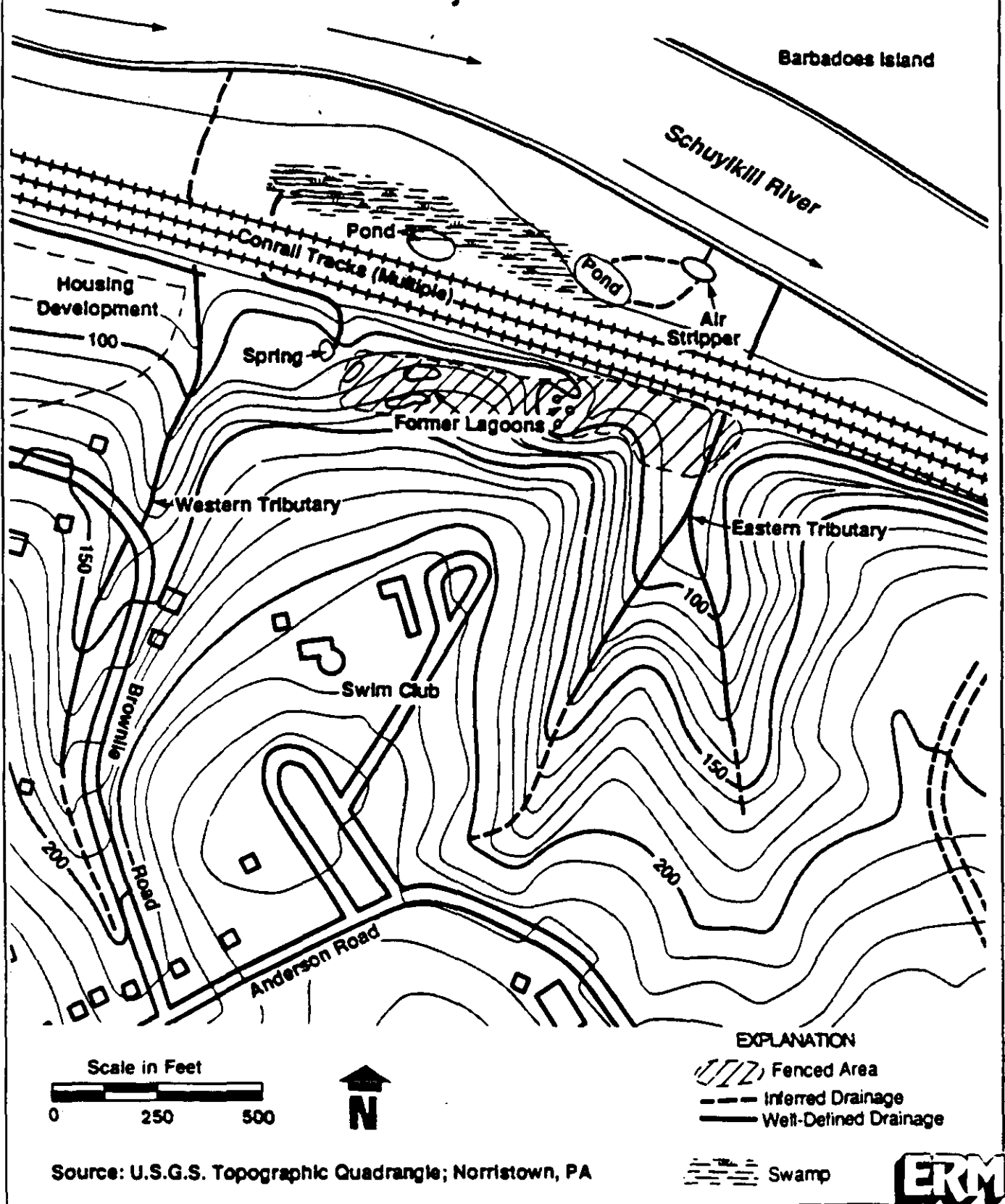
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which originates over 1000 feet south of the site. The upper
reaches of this stream are undeveloped and heavily wooded. The
part of this tributary which crosses the site was re-channelized
as part of the immediate removal actions to diminish the
possibility that contaminants on site would be released into the
stream. The stream, upon reaching the fence which bounds the
On-Site area, flows through several culverts and then passes
beneath the railroad tracks into the floodplain where it
discharges into the Schuylkill River. This tributary is
characterized by minimal discharges which are directly related to
variations in seasonal precipitation.

Another north-south trending tributary flows through a deep
ravine and forms the western boundary of the site. This
tributary receives drainage from the homes in the area and flows
parallel to Brownlie Road approximately 1500 feet. When Brownlie
Road turns westward, the tributary flows beneath it, northward
past the former lagoon area, beneath the railroad tracks and onto
the floodplain. Upon reaching the floodplain, the stream enters
a series of swampy depressions and small ponds, eventually
discharging to the Schuylkill River or infiltrating the soils in
the floodplain. This tributary has been characterized by
variable discharges of relatively low volume. Seasonal
variations and storm-related events directly affect the
tributary's base flow discharge.

A spring originates from a round concrete spring box just below
the westernmost fenced area of the site. The measured discharge
at this spring has varied from 2.5 gpm to 15 gpm. The discharge
rate of this spring appears to be directly related to
precipitation in the area. The spring flows north for less than
100 feet where a drainage ditch running along side the railroad

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tracks diverts flow beneath the railroad tracks onto the floodplain into a swampy area.

The floodplain north of the site contains two seasonal ponds and several swampy areas which are adjacent to the railroad tracks. During prolonged dry periods, the ponds and swamps do not contain standing water and the flow from the western tributary infiltrates the floodplain soils. Under normal conditions, the water entering these areas discharges into the Schuylkill River through numerous small channels.

1.3.10 Other Significant Features

The general vicinity of the RI study area has many features which are indirectly related to the actual investigation. However, these features are meaningful to the total understanding of site conditions. The Conrail railroad tracks, which divide the floodplain from the ridge on which the former lagoon area is located, are likely the oldest man-made features in the area. Their presence is significant because the track construction and right-of-way made the area accessible to vehicles. They also can be a significant source of contamination due to the materials used for the railroad construction and the materials transported by the railroad. The tracks parallel the Schuylkill River and are built on the remnant floodplain of the Schuylkill River.

An 8-inch natural gas pipeline owned by Transcontinental Gas Pipe Line Corporation was reportedly installed while the Tyson's Site was active. The pipeline right-of-ways are well marked and maintained.

Barbadoes Island, located approximately 350 feet beyond the banks of the river, is a maximum of 2000 feet wide and over two miles

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long. Philadelphia Electric Co. (PECO) owns a coal-fired power
plant which was built on Barbadoes Island about fifty years ago.
This plant is currently fully operational, but is only used as a
training facility.

There are three public drinking water supply intakes along the
Schuylkill River between the site and the Delaware River. The
closest downstream intake is about 2,000 feet downstream and
belongs to the PA American Water Company. This plant supplies
drinking water to the Norristown, Pennsylvania, area. The
Philadelphia Water Department intakes at Belmont Avenue and
Queen's Lane are about 12 miles downstream of the study area.

Much of the Schuylkill River flow is controlled by dams of
various designs. There is a small overflow dam approximately
2000 feet downstream from the site. The PA American Water
Company intake is upstream of this dam.

A residential subdivision of 58 single family homes was built
approximately 2000 feet west of the former lagoon area in the
late 1970's and early 1980's. All of the homes in this
development are connected to the Suburban Water Company. Areas
further to the south are also residential. The property
immediately to the south of the former lagoon area is owned by a
local contractor but was previously the Upper Merion Swim Club.
The swimming pools and associated structures are still present on
the property, but not in use. The source of water for the club
during operatin is unknown.

During the early investigations at the site, a leachate
collection system was installed. The design of the system
required re-directing natural run-off to a perforated pipe-sump
collection system in the ditch south of the railroad tracks and

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to an air stripping water treatment plant. Following treatment, water is discharged to an unnamed tributary that empties into the Schuylkill River.

Concurrent with the RI field work conducted by ERM, AT&T subcontractors installed a fiber optics cable along the railroad track right-of-way. The installation of the cable required trenching to a depth of approximately thirty inches.

1.4 Comparison of Organic Compounds Detected in On-Site and Off-Site Samples (ERM, 1986)

An extensive data base exists for the organic compounds and inorganic constituents in the former lagoon area. This includes analysis of subsurface and surface soil samples obtained during the On-Site RI, the Woodward Clyde Consultants Supplemental Soil Investigation, and the SRW investigation of the area west of the former lagoon area. Surface and subsurface soil and sediment and surface water samples from several of the Off-Site areas were also collected during the On-Site RI.

Table 1-2 (4-20 of the Off-Site RI) is a comparison of the organic compounds detected in the former lagoon areas during the above investigations and the organic compounds detected in the Off-Site Operable Units during the On-Site RI and the Off-Site Operable Unit RI. A broad suite of similar organic compounds were detected in both the former lagoon areas and the various Off-Site Operable Units. However, it is also quite obvious from Table 1-2 that the PAHs, PCBs, and pesticides detected during the various investigations did not originate from the former lagoons.

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COMPOUND	ON-SITE ORGANIC COMPOUNDS	EPA OFF-SITE INVESTIGATION	OFF-SITE INVESTIGATION OPERABLE UNITS					SEEP	SEEP AQUIFER
			HILLSIDE	RAILROAD	FLOODPLAIN WATER**	SOILS	SEDIMENT		
2-CHLOROPHENOL	X								
2,4-DIMETHYLPHENOL	X		X						
PHENOL	X	X	X						X
2-METHYLPHENOL	X	X	X			X		X	X
4-METHYLPHENOL	X	X	X			X	X	X	
BENZENE	X	X			X	X	X		X
CHLOROBENZENE	X	X		X	X	X	X		X
ETHYLBENZENE	X	X		X	X	X	X		X
1,2,4-TRICHLOROBENZENE	X	X		X	X	X	X		X
1,3-DICHLOROBENZENE	X	X		X		X			
1,2-DICHLOROBENZENE	X	X		X		X			
1,4-DICHLOROBENZENE	X	X		X	X	X			
NITROBENZENE	X	X							X
NAPHTHALENE	X	X	X	X	X	X	X		
2-METHYLNAPHTHALENE	X	X	X	X	X	X	X		
2-CHLORONAPHTHALENE	X	X	X	X	X	X	X		
N-NITROSODIPHENYLAMINE	X	X		X					
BIS(2-ETHYLHEXYL)PHTHALATE	X	X			X	X			
DI-N-BUTYL PHTHALATE	X	X				X			
DI-N-OCTYL PHTHALATE	X	X				X			
DIETHYL PHTHALATE	X	X				X			
BUTYL BENZYL PHTHALATE	X								
ANILINE	X								
METHYLENE CHLORIDE	X	X							
FLUOROTRICHLOROMETHANE	X								
TETRACHLOROETHENE	X	X	X	X	X	X	X		X
TOLUENE	X	X			X	X	X		X
TRICHLOROETHENE	X	X	X		X	X	X		X
TOTAL XYLENE	X	X			X	X	X	X	X
1,1-DICHLOROETHANE	X	X			X	X	X		X
CHLOROFORM	X	X		X		X			X
TRANS-1,2-DICHLOROETHENE	X	X		X	X	X			X
TRANS-1,3-DICHLOROPROPENE	X	X							
ACETONE	X	X							X
4-METHYL-2-PENTANONE	X	X				X	X		X
HEPTACHLOR EPOXIDE	X								
1,2,3-TRICHLOROPROPANE	X	X		X	X	X	X	X		X
2-BUTANONE	X						X		
1,1,2,2-TETRACHLOROETHANE	X				X				
VINYL CHLORIDE	X								
ALDRIN #	X			X					X
4,4'-DDE #	X	X				X			
4,4'-DDD #	X	X				X		X	
4,4'-DDT #	X	X	X					X	
ENDOSULFAN I #	X		X						X
ENDOSULFAN II #	X				X				X
ENDOSULFAN SULFATE #	X		X						X
HEPTACHLOR #	X								
ALPHA-BHC #	X								
GAMMA-BHC-LINDANE #	X								X
BETA- BHC #	X	X				X			X
PCB (1254,1260) #	X	X							
DIELDRIN #	X	X							X
DELTA BHC #	X								

THE FOLLOWING COMPOUNDS WERE DETECTED IN THE OFF-SITE OPERABLE UNITS ONLY AND THUS ARE NOT RELATED TO THE TYSON'S SUPERFUND SITE.

COMPOUND									
CARBON DISULFIDE *	TID (NOT DETECTED)								
2-HEXANONE *	TID (NOT DETECTED)								
1,1,1-TRICHLOROETHANE				X					
CIS-1,3-DICHLOROPROPENE									X
1,2-DICHLOROPROPANE		X							X
DIBENZOFURAN *		X(TID)	X	X	X				
BENZOIC ACID		X(TID)							X
N-NITROSODI-N-PROPYLAMINE		X							
CHLORDANE		X							
FLUORANTHENE		X	X	X	X	X	X	X	
ISOPHORONE		X							
ACENAPHTHENE		X				X	X		
ACENAPHTHYLENE		X				X	X		
PYRENE		X	X	X	X	X	X	X	
PHENANTHRENE		X	X	X	X	X	X	X	
BENZO(A)ANTHRACENE		X	X	X	X	X	X	X	
BENZO(B)FLUORANTHENE		X	X##	X##	X##	X##	X##	X##	
CHRYSENE		X	X	X	X	X	X	X	
ANTHRACENE		X			X	X	X		
BENZO(A)PYRENE		X		X	X	X	X	X	
BENZO(K)FLUORANTHENE		X				X			
BENZO(GHI)PERYLENE		X			X	X	X	X	
FLUORENE		X			X	X	X		
DIBENZO(A,H)ANTHRACENE		X				X	X		
INDENO(1,2,3-CD)PYRENE		X			X	X	X	X	

* NEL COMPOUND IN EPA INVESTIGATION BUT TENATIVELY IDENTIFIED COMPOUND IN EPA INVESTIGATION

TID - TENATIVELY IDENTIFIED COMPOUND

** Includes surface water samples from the unionlied river and ground water
samples from the EPA wells completed in the uncontaminated deposits on the floodplain.

Detected in background upgradient samples only or at higher concentrations upgradient
than on-site. These compounds are not related to the Tyson's Superfund Site.

Includes both benzo(b)fluoranthene and benzo(k)fluoranthene

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Possible sources of the PAHs to the Off-Site Operable Units include the following:

- coal fines washed downriver from coal crushing/washing and storage operations along the northern reaches of the river;

- burning of construction materials;

- bottom ash used as fill material for the railroad ballast;

- materials used for maintenance and construction of the railroad;

- spills of coal, coal related products, and chemicals during the transport of these materials via the railroad;

- fly ash and gaseous emissions from the coal fired generating station on Barbadoes Island.

1.5 Summary of Introduction

The Tyson's Site has been separated into On-Site and Off-Site Areas by EPA, with the Off-Site area comprised of five separate Operable Units. This EA will evaluate all five of the Off-Site Units.

Many contaminants are present in both the On-Site and Off-Site areas and thus appear to be related to past site activities. However, many of the PAH compounds, PCBs, and pesticides are

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present only in the Off-Site Operable Units, and their absence on-site indicates that sources other than Tyson's may be responsible. Therefore these compounds have not been included in this Endangerment Assessment.

SECTION

2

SECTION 2

METHODOLOGY

2.1 EPA's Endangerment Assessment Process for CERCLA Sites

This section provides a broad overview of the CERCLA Endangerment Assessment (EA) process. The discussion is not intended to be a comprehensive guide to preparing risk assessments. EPA has proposed guidelines for the preparation of EAs in the Endangerment Assessment Handbook (US EPA, 1985a), Superfund Public Health Evaluation Manual (US EPA, 1985b), Exposure Assessment Handbook (US EPA, 1986a), and Toxicology Handbook (US EPA, 1986b).

An EA is normally conducted after the completion of a Remedial Investigation (RI). The RI determines the nature and extent of contamination at a site, and its results form the data base on which potential exposures can be determined and risks assessed. In addition, the RI defines whether or not the present conditions at the site are at steady state.

There are four evaluations which must be completed in a CERCLA EA:

1.   Identification of indicator chemicals, which are used to represent carcinogenic and noncarcinogenic risk at the site;

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2. <u>Exposure evaluation</u>, which includes the calculation of doses to potentially exposed populations;

3. <u>Toxicity evaluation</u> of the potential carcinogenicity of site indicator chemicals, noncarcinogenic effects and development of environmental standards; and

4. <u>Characterization of the risks</u> to a population caused by exposure to each indicator chemical.

2.2 Indicator Chemicals

For the purpose of endangerment assessment, indicator chemicals are selected on a site-specific basis. These are generally the compounds which are most prevalent and provide a representative analysis of risk for the site.

The selection and ranking of indicator chemicals follows the procedure outlined in the draft Superfund Public Health Evaluation Manual (US EPA, 1985b). As part of the indicator chemical selection process, toxicological information about each chemical was compiled using Appendix C of the Draft Superfund Public Health Evaluation Manual (US EPA, 1985b). A range and representative concentration for each chemical was calculated for each appropriate medium. This information includes:

1. toxicologic class potential carcinogens (PC) or noncarcinogens (NC);

2. the severity-of-effect ratings value for non-carcinogens;

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3.   the weight-of-evidence ratings for carcinogens; and

4.   toxicity constants for the various environmental media.

Data used in the selection of indicator chemicals were subjected to comprehensive quality assurance and quality control review. Lancaster Laboratory, Inc. and CompuChem Laboratories, Inc. analyzed the samples from the Tyson's Site Off-Site Operable Unit RI. ERM's quality control and quality assurance procedures, including chain of custody documentation, split samples, replicate analyses, sample spiking with an internal standard, routine instrument calibration, methodology (extraction) blanks, adherence to recommended sample holding times and storage temperatures, were also implemented.

The site related chemicals identified at the Tyson's Off-Site Units were subdivided into potential carcinogens and noncarcinogens. An indicator score (IS), which is the product of the chemical concentration and the toxicity constant (CT), was calculated for each medium and then summed to yield a total indicator score per chemical. The chemicals were then ranked numerically based upon decreasing indicator scores. The top-scoring compounds (based on IS values) were then re-evaluated based upon water solubility, vapor pressure, Henry's law constant, and octanol-water partition coefficient (K_{OC}) to determine the final indicator chemicals. This re-evaluation has a direct relationship to the IS value but selectively eliminates those compounds which are degradation products, have similar physical or chemical properties, or have comparable half-lives in the various environmental media.

2.3 Exposure Evaluation

The purpose of an exposure evaluation is to determine the possible intake of each indicator chemical by a potentially exposed population. The modes of contaminant transport, leading from the sources on the site to a point of exposure, are defined. Potential exposure "scenarios" from contaminated soils are shown on Plate 1 (Appendix AA). Concentrations of the indicator chemicals are determined in each medium with which a population may be exposed (i.e., exposure point concentration). A potentially exposed population is then defined and possible exposure doses are determined. Finally, the possible intake resulting from the potential exposure is calculated.

The sources of contamination at the site are given in the RI. The exposure evaluation considers the migration of contaminants from the site to potentially exposed populations by:

- Evaluating fate and transport process for the indicator chemicals;

- Establishing exposure scenarios for each medium;

- Determining possible exposures to potentially affected populations; and,

- Calculating doses and resultant intakes.

(

2.3.1 Evaluating Fate and Transport Processes for the Indicator Chemcials

The first step in the analysis of exposure is to evaluate the fate and transport processes for the indicator chemicals in a qualitative manner. This is done so that the potential for releases from sources of contamination can be considered in the exposure analysis. This analysis can also identify any significant inter-media transport routes that may need to be evaluated in detail later, in fate and transport modeling. Examples of the fate and transport processes of chemicals in the terrestrial, atmospheric, and aquatic environments are presented in Figures 2-1 and 2-2.

Examples of the environmental fates of the indicator chemicals include sorption by soils and sediments, volatilization into the atmosphere, photochemical degradation, and bioaccumulation. Physical and chemical constants such as solubility and octanol-water partition coefficients are tabulated so that their importance in affecting fate and mobility of the contaminants can be evaluated.

2.3.2 Established Exposure Scenarios for Each Medium

An exposure scenario qualitatively establishes the connection between a source of a contaminant through one or more environmental media to a human population. The mode of exposure

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Figure 2-1
Fate and Transport Processes of Chemicals in the
Terrestrial and Atmospheric Environment

Transport From
Distant Sources
(Background)

Area-Source
Atmospheric
Emissions
(Fugitive Dust
Emissions,
Volatilization)

Point-Source
Atmospheric
Emissions

Precipitation and
Fugitive Dust
Emissions

Wet and Dry
Fallout

Plant Uptake

(See Figure 2-2)

Ground Water
Recharge

Sorption to Soils

Oxidation-Reduction

Outfall

Point-Source
Discharge

Ion Exchange

Percolation and/or Leaching to
Deep Soils and Ground Water

Biodegradation
and/or Bioconcentration

Lagoon

Point-Source
Surface Water
Discharges

Waste Disposal Site

ERM

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Figure 2-2
Fate and Transport Processes of Chemicals in the
Aquatic Environment
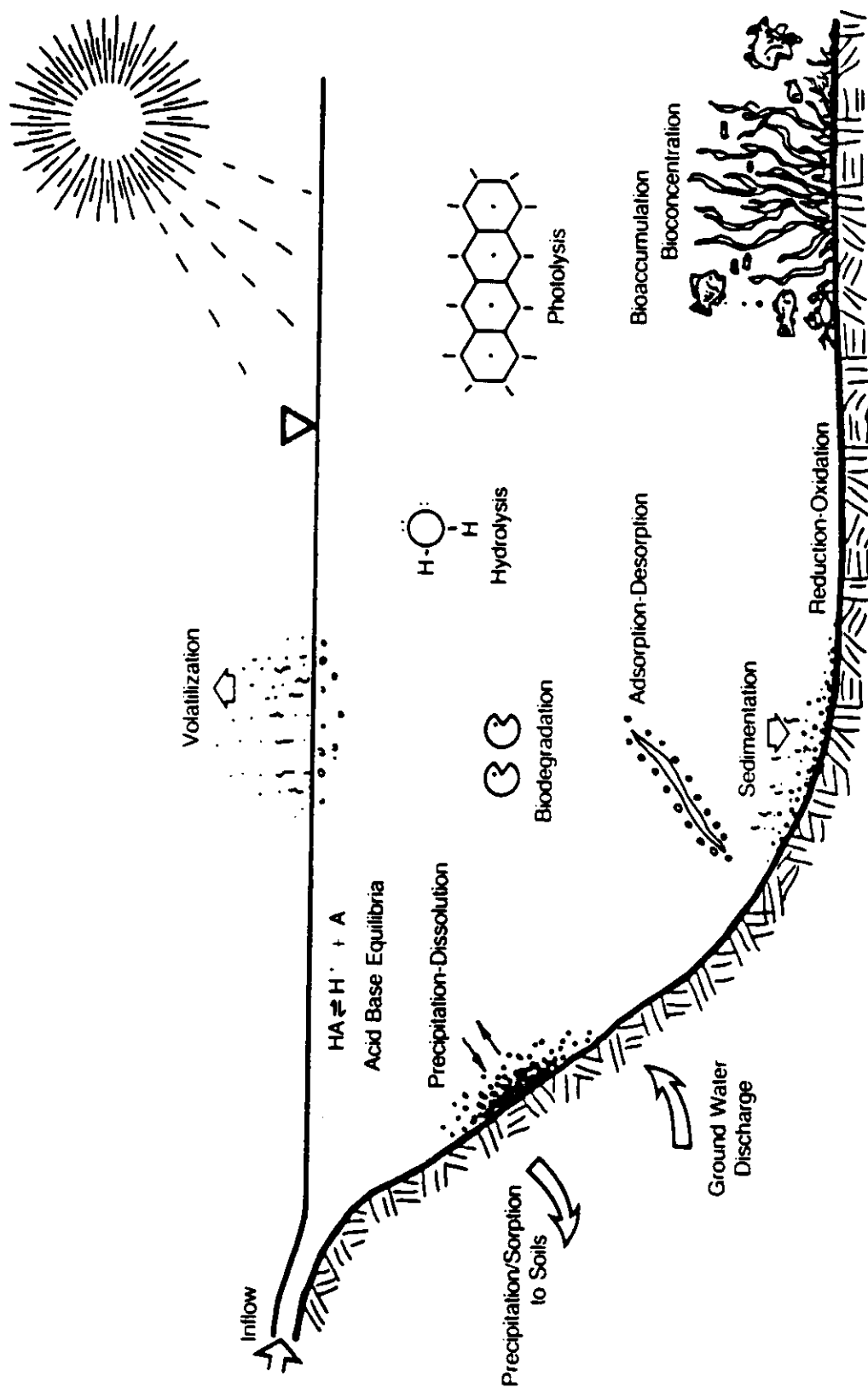
to the population, such as inhalation, ingestion, or dermal contact, is identified as part of the exposure scenarios. Exposure scenarios are determined by integrating information from the RI with knowledge about potentially exposed populations and their likely behavior.

2.3.3 Determining Exposures to Potentially Affected Populations

The next step is the quantitative determination of the exposure concentrations at the potential points of contact with human populations. This step may be quite complicated: it requires knowledge of the contaminant source and its behavior in, and effect on, the environment between the site and any potentially exposed populations. The exposed populations for each medium may also be different, as would be the case if the direction of ground water flow were opposite to that of the prevailing wind.

If the transporting medium can be treated as steady-state, monitoring data may be used to quantify exposure concentrations. If no data are available or if transient, increasing concentrations are suspected, models may be used to predict concentrations.

Ground water contaminant transport through advection and dispersion is normally described in the RI. Transport in such other media as surface water and the atmosphere are not normally evaluated in the RI, and modeling assessments are often required to determine exposures. Many factors, including the fate processes reviewed previously, are considered when selecting the most appropriate model.

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2.3.4 Calculation of Doses to and Possible Intakes by Potentially Exposed Populations

Once exposure concentrations in all media have been determined, the resultant doses and intakes to potentially exposed populations are calculated. Dose is defined as the amount of chemical contacting body boundaries (skin, lungs, or gastrointestinal tract), and intake is the amount of chemical absorbed by the body. To calculate dose and intake several factors must be considered:

-   the amount of contaminated medium that contacts internal or external body surface during each exposure event;

-   the amount of contaminant absorbed during each exposure event; and,

-   the frequency of each exposure event.

Doses and intakes are normally calculated together since they are very similar. Short-term (maximum) and long-term (average) doses are calculated in the same manner. First, for each exposure pathway under consideration, a dose per event is developed. This value quantifies the amount of contaminant contacted during each exposure event. "Event" may have different meanings depending on the nature of the scenario under consideration (e.g., each day's inhalation of contaminated air constitutes an inhalation exposure event). The quantity of contaminant absorbed per event (intake) is calculated by considering the concentration of contaminant in the medium in which exposure occurs, the rate of contact with the medium (inhalation rate, ingestion rate, etc.), and the duration of each event.

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Event-based intake values are converted to final intake values by
multiplying the dose per event by the frequency of exposure
events over the time frame being considered. Subchronic
(short-term) exposure is based on the number of exposure events
that occur during the short-term time frame using maximum
contaminant concentrations in the media to define dosage. It is
intended to represent a 10- to 90-day exposure. Chronic
(long-term) exposures are based on the number of events that
occur within an assumed 70-year lifetime using average
contaminant concentrations in the media to define dosage.

Estimates of daily intakes of contaminants are necessary to
assess risk. Daily intake estimates are expressed in terms of
mass of contaminant per unit of body mass per day. They are
derived by dividing daily exposures by an appropriate average
body mass; a 70 kg adult, for example.

In the Tyson's Site Off-Site Operable Unit assessment, three
routes of exposure are applicable: ingestion of contaminants;
inhalation of volatilized contaminants; and dermal exposure to
contaminants. Calculations were made for each exposure
mechanism according to the Superfund Exposure Manual (US
EPA,1986a).

Both subchronic and chronic intakes were calculated. The
Subchronic Daily Intake (SDI) is the possible human intake of a
chemical averaged over a short time period, and is calculated by
multiplying peak concentrations by human intake and body weight
factors. It is used for subchronic risk characterization.

SDIs and Chronic Daily Intakes (CDIs) were calculated by
adjusting the short-term and long-term doses respectively to

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account for the amount of contaminant that might be absorbed by the body. The resultant intakes were then used in the risk characterization process. For carcinogens, the CDI values are used to assess carcinogenic risk and the SDIs are used to examine acute effects. For noncarcinogens the intakes are used to evaluate acute and chronic effects.

2.3.4.1 Inhalation Exposure

Potential inhalation intakes are estimated based on the number of hours in each event, the inhalation rate of the exposed individual during the event, and the concentration of contaminant in the air breathed. The formula for calculating event-based dosage is:

$$IEX = D \times I \times C \times RF$$

where

IEX = estimated inhalation intake (mass of contaminant per event)

D = duration of an exposure event (hours per event)

I = average inhalation rate of exposed persons (cubic meters per hour)

C = contaminant air concentration throughout the exposure period (milligrams per cubic meter of contaminated air)

RF= retention factor of inhaled compound, i.e., the fraction of the inhaled concentration that is absorbed into the bloodstream (assumed to be an average of 0.50 for most compounds)

Subchronic (short-term) exposure resulting from inhalation is calculated using the maximum contaminant air concentration. Chronic (long-term) exposure is based on the average concentration.

2.3.4.2 Dermal Exposure

Dermal intake is determined by the concentration of hazardous substance in a contaminated medium that is contacted, the body surface area contacted, the duration of the contact and the flux or absorbed fraction. For exposure to contaminated water, dermal intake per event is calculated as follows:

$$DEX = D \times A \times C \times Flux$$

where

DEX = estimated dermal intake per event (mass of contaminant per event)

D = duration of an exposure event (hours per event)

A = skin surface area available for contact (cm^2)

C = contaminant concentration in water (weight fraction)

Flux = flux rate of water across skin ($mass/cm^2/hr$).

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For exposure to contaminated soil, dermal intake per event is calculated as:

$$DEX = WF \times A \times DA \times \text{absorbed fraction}$$

where

WF = weight fraction of chemical substance in soil (unitless)

AV = skin surface area exposed per event (cm^2/event)

DA = dust adherence (mg/cm^2)

Absorbed Fraction = the fraction of the chemical on the skin that is absorbed.

Possible subchronic intake resulting from each dermal exposure event is calculated using the maximum (short-term) contaminant concentrations in water. Chronic intake is based on average (long-term) contaminant concentrations.

2.3.4.3 Ingestion Exposure

Potential intake resulting from ingestion of soil or water borne contaminants is determined by multiplying the concentration of the contaminant in the soil or water by the amount of soil or water ingested per day and the degree of absorption (assumed to be one hundred percent).

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2.4 Toxicity Evaluation

The selected indicator chemicals are subjected to toxicity
evaluation to identify applicable and relevant standards and to
develop a data base against which exposure point intakes can be
compared during the risk characterization evaluation. This
evaluation includes the consideration of experimental studies
using mammals and aquatic non-mammalian species (where
available), as well as relevant standards for humans.

This evaluation presents summaries of health effects data,
pharmacokinetics and metabolism, toxic and carcinogenic effects,
and applicable and relevant standards available for the three
indicator chemicals. Because of its major impact on the risk
evaluation, the procedures used for classifying animal and human
carcinogens by both the EPA and the International Agency for
Research on Cancer of the World Health Organization (IARC), and
the attendant uncertainties, will be presented.

Evaluations of carcinogenicity basically involve two steps: (1)
the identification of potential carcinogens among the
contaminants present at the site and (2) the quantitative
determination of their carcinogenic potency.

Evidence of possible carcinogenicity in humans comes primarily
from long-term animal tests and epidemiological investigations.
Results from these studies are supplemented with information from
short-term tests, pharmacokinetic studies, comparative metabolism
studies, structural-activity relationships, and other relevant
information sources.

For judging the qualitative evidence of carcinogenicity, EPA as
well as the IARC have adopted a policy of "weight-of-evidence",

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meaning that the quality and adequacy of all relevant data on responses induced by a possible carcinogen using different procedures will be considered. There are three major steps in determining the weight-of-evidence for carcinogenicity:

1. Characterization of the evidence from human studies and from animal studies individually,

2. Combination of the two types of studies into a final indication of overall weight-of-evidence for human carcinogenicity, and

3. Evaluation of all supportive information to determine if the overall weight-of-evidence should be modified.

Further details concerning the classification systems of the EPA and the IARC, and on the use of this data in the risk assessment process, are presented in Appendix A.

The second phase in carcinogen assessment involves the quantification of risk. Experimental studies of carcinogenic effects that utilize the low exposure levels usually encountered in the environment usually are not feasible. Therefore, various mathematical models have to be used for extrapolation from the high doses used in animal bioassays down to the dosages involved with exposure to ambient environmental concentrations. Since the resolution power of animal studies, for example, are not adequate for precise elaboration of the dose-response curve, extrapolating from a high dose to a low dose introduces a level of uncertainty which may amount to orders of magnitude. Given the recognized differences in carcinogenic response between species, and between strains of the same species, it is clear that additional uncertainties will be introduced when quantitative

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extrapolations, as from rodents to humans, are made. Of the various proposed models for quantitative extrapolation, EPA recommends a linearized multistage model: "In the absence of adequate information to the contrary, the linearized multistage model will be employed." (Federal Register, Guidelines for Carcinogen Risk Assessment, September 24, 1986). The linearized multistage model assumes linearity at low doses. Alternative models do not assume a linear relationship and in general are much less conservative. There is no biologically sound basis for choosing one model over another. However, when applied to the same data the various models can produce a wide range of risk estimates; the model recommended by EPA usually produces the highest estimates of risk. Moreover, this model does not provide a best estimate of risk, but rather an upper bound probability that the risk will be less 95% of the time.

2.5 Risk Characterization

The risks to the potentially exposed population from exposure and subsequent intakes of the indicator chemicals are calculated in three tasks:

1. Carcinogenic Risk,

2. Noncarcinogenic Risk, and

3. Comparisons with Environmental Standards.

2.5.1 Carcinogenic Risk

For potential carcinogens, risks are estimated as probabilities. The carcinogenic potency factor, which is the upper 95% confidence limit of the probability of a carcinogenic response per unit intake over a lifetime of exposure, converts estimated CDIs directly to incremental risk values. As discussed in Section 6.6 below, this is the methodology which EPA required us to use. It is not the only methodology and is rather likely to be an upper bound. In general, because only relatively low CDIs are likely to result from environmental exposures, the EPA methodology assumes that the exposure will be in the linear portion of the dose-response curve. Based on this assumption, the slope of the dose-response curve is equivalent to the carcinogenic potency factor, and the risk is directly proportional to the CDI at low levels of exposure. The low-dose carcinogenic risk equation is:

$$\text{Risk} = \text{CDI} \times \text{Carcinogenic potency factor}$$

2.5.2 Noncarcinogenic Risk

The Hazard Index method is used for assessing the overall potential for noncarcinogenic effects posed by multiple chemicals. The Hazard Index calculates a safety margin, a factor by which the acceptable intake exceeds the estimated exposure level. This approach assumes that multiple sub-threshold exposures could result in an adverse effect and that the magnitude of the adverse

effect will be proportional to the sum of the ratios of the sub-threshold exposures to acceptable exposures. This can be expressed as:

Hazard Index = E_1/AL_1 + E_2/AL_2 + ... + E_i/AL_i

where:

E_i = Exposure level (or intake) for the ith contaminant

AL_i = Acceptable level (or intake) for the ith contaminant

For a single contaminant, there may be a potential adverse health effect when the hazard index exceeds unity, though because the "acceptable level" itself incorporates a large margin of safety (safety factor) no toxic effects may occur even if the "acceptable level" is exceeded. For multiple chemical exposures hazard indices, if summed, may result in an overall hazard index that exceeds one even if no single chemical exceeds its acceptable level. However, the assumption of additivity should only be made for compounds that produce the same toxic effect by the same mechanisms of action.

EPA has developed some preliminary information regarding Acceptable Intakes for Subchronic Exposures (AISs) and Acceptable Intakes for Chronic Exposures (AICs)(Mabey,W.R.,et al,1982), and USEPA, 86B. Where these are available, they are used as acceptable levels for subchronic and chronic exposures, respectively.

2.5.3 Comparison with Environmental Standards

In this section the exposure point concentrations of all contaminants are compared to applicable or relevant and appropriate standards as defined by the National Contingency Plan (NCP) and identified in the CERCLA compliance policy memo, which is an appendix to the NCP. At present, EPA considers drinking water maximum contaminant levels (MCLs), national ambient air quality standards (NAAQS), and federally-approved state water quality standards developed under the Clean Water Act to be potentially applicable, or relevant and appropriate ambient concentration requirements.

2.6 Uncertainty

U.S. EPA employs a great deal of conservatism in the process that it proposes to describe human cancer risks. EPA gives animal test evidence stronger weight in determining the strength of evidence that a substance is a human carcinogen than the IARC. In this process, EPA includes benign liver tumors in its estimations. Additionally, EPA suggests the use of the linear multistage dose-response model to predict human cancer risk at low doses. This model is more conservative than other standard dose-response models. It forces linearity on the dose-response curve, even if the experimental data are clearly non-linear, and it uses statistical upper confidence limits on risk rather than most likely estimates. Further, EPA's procedure for interspecies extrapolation uses a very conservative procedure based on relative body surface area rather than body weight. EPA's own Science Advisory Board has frequently recommended in the past that other models be used to illustrate the range of uncertainty

in risk assessment. This issue is discussed in detail in Section 6.6.

EPA is conservative in calculating intakes over a lifetime, for instance, in determining the amount of air that is breathed or water consumed. EPA assumes that substances ingested are completely absorbed through the digestive tract, although it is known that only a fraction of the mass behaves in this way. These combined factors lead to a very high level of conservatism in the overall process with the effect of over-estimating risks. The scenarios developed as part of this assessment for the Tyson's Site Off-Site Operable Unit EA follows this philosophy as well, leading to a probable overall over-estimation of risk.

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SECTION

3

SECTION 3

SECTION 3

INDICATOR CHEMICALS

3.1 Selection of Contaminants of Concern

The numerous contaminants identified in the study area comprise a diverse group of compounds with widely disparate physicochemical, environmental, and toxicological properties. The extent of contamination study also revealed wide variations in concentration and occurrence. Some contaminants, accordingly, represent a greater potential risk to human health or the environment than others because of differences in inherent toxicity, capacity to migrate to receptors, and likelihood of exposure of receptors to concentrations sufficient to pose risk of injury. It is neither necessary nor practical to evaluate each contaminant in terms of the many parameters that determine transport, exposure, and attendant health or environmental risk in order to effectively address endangerment. Rather, a selective identification of contaminants of concern is undertaken to focus this effort on a limited set of compounds which adequately represent the major hazards associated with a particular site and its unique conditions. Selection of indicator chemicals was performed in accordance to procedures outlined in the Endangerment Assessment Handbook and described in detail in the Superfund Public Health Evaluation Manual (U.S. EPA, 1987).

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The first task in the indicator chemical selection process is the development of a preliminary list based entirely on chemical toxicity information and site concentration data. An indicator score (IS) is calculated by multiplying the maximum or average concentrations by a media-specific toxicity constant (US EPA, 1987). The chemicals can then be ranked by their indicator scores. By considering additional factors, such as mobility, frequency of detection, extent of contamination similarity to other related compounds, bioconcentration potential, persistence, etc., a final group of representative compounds of concern is then selected.

3.2 Indicator Chemical Selection

The indicators are chosen to represent the site-related compounds detected in the off-site operable units. Several assumptions were made during review of chemical data for the Tyson's Site Off-Site Operable Units:

(1) Only data collected during the ERM Off-Site RI and data collected outside the fenced area during the Baker/TSA investigation were used during the indicator chemical selection process.

(2) The selection process was limited to Hazardous Substance List (HSL) inorganic constituents and organic compounds. An additional forty peaks ("tentatively identified compounds" (TICs)) are requested by the EPA. These compounds are labeled as tentative since their identification is not made through the use of standards but through the closest match of the mass spectrum with

the mass spectral library. Therefore, "tentatively identified compounds" are not included in the process because concentrations of these compounds are not quantifiable (i.e., estimated values).

(3) The concentration of HSL inorganics in soil samples were compared to the observed background range for inorganics in the Eastern United States (Connor, J. and Shacklette, H., 1975) and to ranges in background soil samples where appropriate. Inorganics whose concentrations detected in soils fell within the background range, were disregarded in the indicator chemical selection process and considered as typical soil concentrations unless they were found in other media at levels above background. Those inorganics found at levels above background concentrations in soil or in other media are considered in the selection process.

(4) Organics removed from consideration were those detected infrequently and/or at very low concentrations.

(5) Although no toxicity constants are available in the Superfund Public Health Evaluation Manual for 1,2,3-trichloropropane, it is evaluated in the indicator chemical selection process due to its concentrations and frequency detected in the Off-Site Operable Units. Regional EPA has classified 1,2,3-trichloropropane as a B2 carcinogen based upon verbal preliminary data at an interim stage of a two-year study being conducted by the National Toxicology Program (NTP). These data are subject to change upon completion of the study and internal and

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external review processes. For the purposes of this
document, ERM treats 1,2,3-trichloropropane as if it
were a carcinogen as requested by EPA.

A range and representative concentration was determined for each
chemical evaluated during the indicator chemical process.
Toxicological information consisting of potential carcinogen (PC)
and/or noncarcinogens (NC) classification, and toxicity constants
for water, soil and air were obtained from Appendix C of the
Superfund Public Health Evaluation Manual (USEPA, 1987). The
worksheets used to develop CT (media concentrations times
toxicity constants) and indicator score (IS) values are presented
in an appendix to this report. Information relative to the
ranking of carcinogens and noncarcinogens is also presented in
Appendix C. The initial short-list indicator chemicals and the
assumptions used to select the final indicator chemicals for the
Tyson's Off-Site Operable Units are given in Table 3-1.

3.3 Discussion of the Classification of Chemicals

A general discussion of the chemical classes and several
representative compounds detected during the Off-Site Operable
Unit RI is presented here to emphasize the need to select
indicator chemicals from each class.

The volatile organics are comprised, _in general_, of halogenated
saturated and unsaturated aliphatic and selected aromatic
compounds. These compounds are generally soluble in water and
have relatively high volatility rates. They are not adsorbed to
soils/sediments or suspended particles and are therefore

TABLE 3-1
JUSTIFICATION FOR INCLUSION OF
CHEMICALS ON THE FINAL INDICATOR CHEMICAL LIST

COMPOUND	JUSTIFICATION	RELATIVE IS RANKING PC	NC
Arsenic	Concentrations in the ground water are at the MCL level	1	11
Chlorobenzene	Concentrations found in all media and in DNAPL		12
Benzene	Known human carcinogen - high concentrations in some ground water samples	5	18
1,2,4-Trichlorobenzene	Detected all media although concentrations were low		13
1,2,3-Trichloropropane	Found in all media, in highest concentrations, and especially in DNAPL	-	
Barium	Unfiltered samples showed high concentrations but several filtered samples showed detectable concentrations above the RMCL		2
Trichloroethylene	Frequently detected in all media	2	4
Xylene	Detected in all media and in DNAPL		15
Toluene	Detected in all media and in DNAPL		17
Ethylbenzene	Detected in all media and in DNAPL		19
Chloroform	Ranked high in indicator chemical process and concentrations above MCL in ground water	3	
1,2-Dichloroethane	Frequency of detection is very low for this compound	5	29
Tetrachloroethylene	Frequently detected in all media	4	21

available for transport to ground water through leaching
processes, to surface water through runoff, and to the atmosphere
through volatilization from soils or surface water (or both).

The most frequently detected volatile organics in the Tyson's
Off-Site Operable Unit were trichloroethylene,
1,2,3-trichloropropane, tetrachloroethylene, toluene,
ethylbenzene and xylenes which are related to past on-site
operations at Tysons.

The semi-volatile organics contain two classes: the base-neutrals
and the acid extractables. Base-neutrals are those compounds
which require basic or neutral pH conditions for extraction from
environmental samples. In general, these compounds are quite
strongly adsorbed to organic matter with which they come into
contact, such as soils/sediments or suspended particles. Because
of this, they are likely to be substantially retarded during
movement of the contaminants through soils and ground water
systems. However, they may be readily transported by surface
runoff during high rainfall events, to the atmosphere as fugitive
dust, and possibly by volatilization from soils and surface
waters to the atmosphere.

The base-neutrals most frequently detected during the Tyson's
Off-Site Operable Unit RI are bis(2-ethylhexyl)phthalate,
di-n-butyl phthalate, and 1,2,4-trichlorobenzene which are
related to past on-site operations at Tysons.

The acid extractables are phenolic compounds (in some cases
listed as cresols) and require acidic pH conditions for their
extraction from environmental samples. Phenolics have moderate
to high water solubilities but extremely low volatilization rates.
As a class, their degree of sorption onto soils/sediments and

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suspended particles is mixed. Phenolic compounds do not readily volatilize, but are susceptible to oxidation/photolysis and biodegradation. Phenol, 2,4-dimethylphenol, and 4-methylphenol which are related to past on-site operations at Tysons were the acid extractables detected during the Off-Site Operable Unit RI.

The inorganic class contains the trace elements and the metals. Fate and transport processes are dependent upon the chemical speciation of the inorganic compound. In general, the inorganics are not soluble in water and do not volatilize. However, these constituents do sorb to soils/sediments or suspended particles, thus limiting their transport to the ground water, surface water and atmosphere. Arsenic, which is related to past on-site operations at Tysons, was the only inorganic constituent detected above the background soil levels during the Off-Site Operable Unit RI. Barium, which was not present above background levels in soil, was present at elevated levels in deep ground water. It is unclear whether this elevation is related to on-site operations at Tysons.

3.3.1 Summary of the Indicator Chemicals

Indicator chemicals were selected for the aforementioned media from the off-site units using the proposed EPA procedures and methods detailed in Section 2.2. Results of this selection process are in Appendix C: Worksheets 1 through 5. Primary consideration in the final selection of indicator chemicals was based upon the levels and frequency with which a compound was detected in all media for the off-site areas. The resulting selected indicator chemicals for use in the Tyson's Off-Site Operable Unit Endangerment Assessment are listed in Table 3-2.

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TABLE 3-2

INDICATOR CHEMICALS FOR THE
OFF-SITE OPERABLE UNITS AT
THE TYSON'S SITE

Arsenic
Chlorobenzene
Benzene
1,2,4-Trichlorobenzene

Xylene
Toluene
Ethylbenzene
Chloroform

Tetrachloroethylene
Trichloroethylene
Barium
1,2,3-Trichloropropane

SECTION

4

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SECTION 4

EXPOSURE ASSESSMENT: EXISTING CONDITIONS

This section evaluates potential exposures to human populations
for the following Tyson's Site Off-Site Operable Units:

- Deep Aquifer (Operable Unit 1)

- Hillside Area (Operable Unit 2)

- Railroad Area (Operable Unit 3)

- Floodplain/Wetlands Area (Operable Unit 4)

- Seep Area (Operable Unit 5)

The exposure assessment is presented for each individual Off-Site
Unit in separate subsections. Information concerning the
processes influencing the fate of the indicator chemicals is
provided in Appendix C.

4.1 Deep Aquifer

The deep aquifer is the connecting unit present under all other
operable units, consisting of sandstone units with random, thin
lenses of shale. The Schuylkill River is a regional discharge
area, and it is expected that the portion of the deep aquifer

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underlying the on-site unit and the other operable units will discharge to the River.

4.1.1 Environmental Fate and Transport

The previous source of contamination to the deep aquifer was the former lagoons. It is known that bulk liquid chemicals were disposed of at Tyson's Site directly into the unlined former lagoons. The lagoons were situated directly upon or within the sandstone bedrock and the sandstone near to the bedrock surface is known to be extensively fractured. It is not known what the levels of liquids were in the lagoons during their operation, but they were almost certainly sufficient to overcome capillary forces and drive dense non-aqueous phase liquid (DNAPL) chemicals down into the sandstone bedrock. DNAPL penetration would occur primarily through fractures and bedding plane partings in the sandstone but could also penetrate into coarse-grained permeable units in the sandstone, both in the unsaturated and saturated zones. The migration of DNAPL through the sandstone would be controlled by the orientation and interconnection of the fractures, and the orientation and extent of the coarse-grained beds.

Migration through the bedrock would continue until the volume of DNAPL which penetrated the bedrock was completely assimilated into the sandstone as residual along the fractures and within the coarse-grained beds, or the density-induced downward pressure gradient of the DNAPL was diminished to the extent that it would be counter-balanced by capillary resistance or relative permeability of the strata. A complete and detailed discussion of subsurface contamination by DNAPL chemicals at Tyson's Site is provided in Feenstra and Cherry (1986) and the RI Report, and will not be repeated here. There are currently three sources of

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ground water contamination in the deep aquifer: (1) the DNAPL within the bedrock aquifer, (2) DNAPL present in unsaturated bedrock immediately below the lagoons, and (3) the contaminated soils in the former lagoons. Because of the estimated quantities of DNAPL in the bedrock and the bedrock aquifer (Feenstra and Cherry, 1986), the present contribution of the soils in the former lagoon area to the ground water contamination is negligible. However, dissolved contaminants released from the DNAPL are present in ground water which subsequently discharges to the Schuylkill River. This discharge is estimated to occur at a the overall flow rate of approximately 21,700 ft^3/day, with a subsequent calculated discharge of approximately 14,900 kg/yr of 1,2,3-TCP (ERM, 1987).

4.1.2 Potential Exposure Scenarios

The sources of contamination, the possible transport mechanisms, and the potential modes of exposure to humans from contaminants present in ground water in the deep aquifer are represented in Plate 2 (Appendix D). The following exposure scenarios have been identified:

1)   Human exposure to contaminated ground water used for drinking water purposes. A careful review of the geology of the site and the lower member of the Stockton Formation showed that there is no surficial aquifer in the unconsolidated material on top of the bedrock aquifer (ERM, 1987). The unconsolidated material at the site appears to be the fill material used to backfill the lagoons at the site. Even if there is any naturally occurring unconsolidated material on top of the bedrock, a public water-supply well cannot be developed in it because the required

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minimum casing lengths (40 feet in sandstone and 5 to 10 feet below the pumping level in sand; PA DER Manual) would prohibit its use as a source of water supply even if the Tyson's Lagoon site was not used for any waste disposal purposes. Current regulations also do not permit the construction of public water-supply wells in the floodways. Due to wetland and flood-plain area and availability of public water supply, no housing development with on-site water supply would be anticipated in the area between the railroad tracks and the river. Therefore, potential use of this water for drinking purposes via a well is not possible due to the applicable restrictions on ground water development in the hillside, railroad, seep and floodplain areas.

2) Industrial and/or agricultural use of contaminated ground water. The presence of the hillside, railroad, and floodplain between the site and the downgradient discharge point for ground water effectively prevents direct use of this water for industrial or agricultural purposes via a well.

3) Human exposure to contaminated surface water used for drinking water purposes. As discussed in the Remedial Investigation, direct discharge of ground water to the Schuylkill river is estimated to occur at the overall flow rate of approximately 21,700 ft^3/day. This scenario will be evaluated further.

4) Direct exposure to contaminated sediments and/or surface water during recreational activities. Direct exposure to contaminated sediments is not expected to occur, due to the inaccessibility of these sediments to

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humans; sediments were found to be extremely localized and limited during the RI. Dermal contact with contaminated surface water during recreational activities is a possible source of exposure and will be evaluated further.

5)   In bioaccumulation studies described in the RI, Section 4, using TCP as the site-specific indicator chemical, no TCP was detected. Therefore, consumption of fish and shellfish was eliminated as a pathway of exposure in this risk assessment.

In summary, exposures to contaminated surface water through recreational use and use for household and drinking water purposes are considered.

4.1.3 Exposures to Potentially Affected Populations

The overwhelming majority of exposure of human populations to contaminants in surface water will occur via the household use and/or drinking water pathway. Dermal exposure to contaminants in surface water during recreational activities conventionally results in intakes more than an order of magnitude less than those incurred while showering with water of equivalent quality. Also, ingestion exposure in these circumstances is likely to be several orders of magnitude less than those resulting from the use of water of equivalent quality as drinking water. As such, recreational exposure will not be evaluated further.

There are three water supply intakes on the Schuylkill River between the Tyson's Site and the Delaware River; the Pennsylvania American Water Company intake at Norristown, and the two

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Philadelphia Suburban Water Authority (PSWA) intakes at the Belmont and Queen's Lane Treatment Plants. It is assumed that water treatment will have a minimal effect on indicator chemical concentrations at the three plants. Treatment generally consists of sand filtration and, rarely, activated carbon filtration (i.e., only when complaints of odors or taste necessitate). Because no substantial blending of water from other sources occurs, the sample data from the Schuylkill River are assumed to be potentially representative of the levels that may occur in the distribution system and at the consumers' taps (no tap water samples have been collected). Concentrations used for the calculation of intakes were obtained from ERM data presented in Appendix E.

Adults and children are potentially exposed populations.

4.1.4 Calculation of Doses and Resultant Intakes

Total daily exposure to compounds found in household and/or drinking water is estimated by considering four sources of exposure:

1) ingestion of drinking water,
2) inhalation while showering or bathing,
3) dermal absorption while showering or bathing, and
4) inhalation of household air (accounting for release of contaminants into air via dish washing, clothes washing, etc.).

The methodology used for calculation of doses and intakes is presented in Section 2. Table 4-1 lists some assumptions utilized in estimating exposure, with an indication of their acceptance and/or conservatism.

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Table 4-1

Assumptions Made in the Estimation of Potential Exposure Due To Deep Aquifer

Source of Exposure	Assumptions	Acceptance/Conservatism
Ingestion	Daily consumption by adults of 2 liters of drinking water from the household tap; children consume 1 liter.	Widely accepted as a reasonable, if conservative, estimate.
Dermal Absorption via showering/bathing	Assumption is that VOCs pass the dermal layer in proportion to the flux of water, and that 100% is absorbed.	Reality is unknown.
Inhalation via showering/bathing	- Extent of contaminant volatilization is 100%. - A prolonged duration of showering (probable range 3 to 30 minutes). A value of 20 minutes is assumed. - 200 liters of water use for a 20 minute shower - Standard breathing rate of .83 m3/hr for adults, .44 m3/hr for a pre-teen, .26 m3/hr for an infant/toddler - 50% of the inhaled concentration is absorbed into the bloodstream - Shower stall is a largely confined space with a volume of 3 m3 - Bathroom space is 10 m3, unventilated - Bathroom is occupied for 10 minutes after completion of shower	For some volatile organics, the conservative nature of the assumption that 100% of the compound is volatilized would not introduce a large margin of error. For TCP, however, the assumption of total volatilization may be inappropriate.
Inhalation of household air	- Magnitude of transfer of contaminant from water to household air is assumed to be similar to that of radon (concentration in household air is approximately 0.0001 times the concentration of contaminant in water) - 50% of the inhaled concentration is absorbed into the bloodstream	Analogy to transfer of radon from water to household air presents probable overestimate.

Note: Assumptions for both the chronic and subchronic exposure scenarios are the same; exposure varies only as a function of the concentration of the contaminant in the media of concern.

The doses and resultant intakes from these routes of exposure are presented in Table 4-2.

4.2 Hillside Area

The Hillside Area is bounded on the north side by the On-Site Unit (Figure 1-1), on the south side by the Railroad Area (Operable Unit 3), on the western edge by the Seep Area (Operable Unit 5), and on the eastern edge by the On-Site Unit and the Railroad Area. Vertically it consists of one to two feet of surface soils on top of the bedrock and the surface of the exposed bedrock.

4.2.1 Environmental Fate and Transport

The major source of contamination affecting the Hillside Area appears to have been the former lagoons. This could have resulted by any one of a combination of:

- direct overflow from the lagoons,

- contaminated overland flow during high rainfall events, or

- discharge of contaminated ground water through the exposed bedrock in the Hillside Area.

Residual contamination present in the Hillside Area soils themselves is considered to be the only remaining source of contamination in this area.

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Table 4-2
Calculation of Potential Intakes
Due To Deep Aquifer

Subchronic Intakes (mg/kg/day)

Indicator Chemical	Concentration * (mg/l)	Ingestion of Drinking Water	Dermal Absorption via Showering	Inhalation via Showering	Inhalation of Household Air
Arsenic		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Barium		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Benzene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chlorobenzene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chloroform		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Ethylbenzene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Tetrachloroethene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Toluene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
1,2,4-Trichlorobenzene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Trichloroethene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
1,2,3-Trichloropropane	3.50E-04	1.00E-05	1.26E-06	2.94E-05	2.49E-06
Xylenes	7.00E-03	2.00E-04	2.56E-07	5.88E-04	4.97E-05

Chronic Intakes (mg/kg/day)

Indicator Chemical	Concentration * (mg/l)	Ingestion of Drinking Water	Dermal Absorption via Showering	Inhalation via Showering	Inhalation of Household Air
Arsenic		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Barium		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Benzene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chlorobenzene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chloroform		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Ethylbenzene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Tetrachloroethene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Toluene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
1,2,4-Trichlorobenzene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Trichloroethene		0.00E+00	0.00E+00	0.00E+00	0.00E+00
1,2,3-Trichloropropane	2.23E-04	6.36E-06	8.14E-09	1.87E-05	1.58E-06
Xylenes	3.53E-04	1.01E-05	1.29E-08	2.96E-05	2.51E-06

Note: Blank spaces indicate none detected

* Concentrations used for the calculation of intakes are those obtained during RI sampling of the Schuylkill River. The Schuylkill River is assumed to be the exposure point for contaminated groundwater (Deep Aquifer).

From the contaminated surface soils on the Hillside Area, five
modes of environmental transport of contaminants are possible: 1)
dissolution into runoff, 2) wind induced soil erosion resulting
in fugitive dust emissions, 3) volatilization of contaminants,
4) bioaccumulation, and 5) leaching of contaminants with
discharge to the bedrock flow system.

4.2.2 Potential Exposure Scenarios

The possible transport mechanisms of residual contaminants in the
Hillside soils and potential modes of exposure of humans from
contaminants present in this Unit are presented in Plate 3
(Appendix D). The following exposure scenarios could result from
these transport pathways:

1) <u>Human Exposure to Contaminated Runoff</u> - Runoff occurs
   in the form of overland flow after heavy precipitation
   events on the Hillside. However, the leachate
   collection system at the base of the Hillside (Figure
   1-1), located underneath the drainage ditch on the
   south side of the railroad tracks, almost immediately
   collects this runoff which is subsequently treated in
   the air stripper. This treatment prevents any further
   direct transport of contaminants in this manner.
   Because of the intermittent nature of this mode of
   transport and its almost immediate collection, exposure
   scenarios associated with runoff are not considered
   further.

2) <u>Inhalation of Fugitive Dust</u> - The presence of dense,
   perennial vegetative cover on the Hillside soils
   effectively reduces the probability of soil erosion and

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suspension of inhalable soil particles is unlikely to occur. This scenario is not evaluated further.

3) **Inhalation of Volatilized Contaminants from Surface Soils** - The low concentration of volatile organics in the Hillside surface soils, plus the limited thickness of these soils, indicates that the potential for substantial volatilization of contaminants from these soils has been previously exhausted. This scenario will not be examined further.

4) **Contact with Contaminated Soil** - The Hillside Area is expected to be accessible to children of 6-12 years of age for play activities. This exposure scenario will be evaluated further.

5) **Ingestion of Contaminated Soil** - This area is not expected to attract children exhibiting pica behavior due to the steep gradient and general inaccessibility, and pica behavior will not be evaluated further. However, incidental ingestion of limited amounts of soil during play activities of children 6-12 years of age will be evaluated in conjunction with the dermal contact discussed previously.

6) **Bioaccumulation of Contaminants** - The soils present on the Hillside are extremely shallow, which negates the possibility for food production by humans or wildlife use. Therefore, this transport mode is not considered a viable pathway for further analysis.

7) **Exposure to Contaminated Ground Water** - Soil contaminants could be leached from the Hillside and

transported to the Deep Aquifer (Operable Unit 1).
However, this is not expected to be a significant
source of contamination of the Deep Aquifer because of
the relatively small size of the total contaminant mass
present in the Hillside soils compared to the estimates
of contaminant masses in the Deep Aquifer associated
with the DNAPL (Feenstra and Cherry, 1986).

In summary, dermal contact with contaminated soils, with
incidental ingestion of these soils, is evaluated as the
potential exposure scenario.

4.2.3 Exposures to Potentially Affected Populations

Consideration of the exposure scenarios identified in the
previous subsection indicates that exposure of human populations
to the indicator chemicals present at the Hillside Area will
occur only within the Hillside itself, since there is no
appreciable transport of contaminants to receptors outside the
unit. Soil contaminant concentrations were obtained from those
data presented in Appendix E.

Potentially exposed populations identified at this location are
children who reside nearby. Although accessibility to the area
is hampered by steep slopes, the presence of the railroad tracks,
and the existing fence around the On-Site Area, some recreational
use by children is possible. Such use by adults is not expected.

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4.2.4 Calculation of Doses and Resultant Intakes

The exposure scenario identified requires evaluation of the
following route of exposure for the exposed population (i.e.
children):

1)   dermal absorption of contaminants with accompanying
     incidental ingestion.

The methodology used in evaluating these exposures is presented
in Section 2 of this document. Assumptions concerning length of
exposure and parameters used in exposure calculations are
presented in Table 4-3.

Children of 6-12 years of age were selected for evaluation of
exposure from dermal contact, assuming a site visitation
frequency of 150 days per year. It is assumed that children of
2-6 years of age would have some dermal exposure, however, that
exposure would be considered negligible compared to the exposure
from ingestion of soil. Using these assumptions, the calculated
doses and resultant intakes from the dermal route of exposure are
presented in Table 4-4.

4.3 Railroad Area

The Railroad Area is bounded to the north by the Hillside Area
(Operable Unit 2), and to the south by the Floodplain Area
(Operable Unit 4). The eastern and western boundaries of the
Railroad Area are shown in Figure 1-1. Vertically it consists of
the soils and ballast materials overlying bedrock (Figure 1-2),
with depth to bedrock varying from three to twenty-seven feet.

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Table 4-3

Assumptions Made in the Estimation of Exposure Incurred
Hillside Area

Source of Exposure	Assumptions	Acceptance/Conservatism
Dermal absorption of contaminants in soil during play	- Assumes that 10% of the total concentration of contaminants present in soil adhered to hands passes the dermal layer (dermal area varies with age); data on dust adherence to skin is limited, and is assumed to fall in the following range: Commercial potting soil:1.45 mg/cm2 Clay mineral kaolin:2.77 mg/cm2 - Assumes one complete coating event per day; chronic exposure scenario assumes exposure occurs 150 days per year. - Assumes exposure occurs only to children 6 to 12 years of age - Assumes incidental ingestion of 100 mg of soil in conjunction with dermal contact	Dust adherence will depend on a variety of site specific factors; contact frequency will vary widely and will depend on a large number of factors. Approach presents probable overestimate.

Note: Assumptions for both the chronic and subchronic exposure scenarios are the same; exposure varies only as a function of the concentration of the contaminant in the media of concern.

Table 4-4
Calculation of Intakes
Hillside Area

Indicator Chemical	Concentration (mg/kg)	Subchronic Intake (mg/kg/day) Dermal Absorption with Incidental Ingestion via Soil
Arsenic	3.84E+01	2.76E-04
Barium*		
Benzene		0.00E+00
Chlorobenzene		0.00E+00
Chloroform		0.00E+00
Ethylbenzene		0.00E+00
Tetrachloroethene	3.00E-02	2.17E-07
Toluene		0.00E+00
1,2,4-Trichlorobenzene		0.00E+00
Trichloroethene	2.10E-02	1.52E-07
1,2,3-Trichloropropane	2.50E-01	1.81E-06
Xylenes		0.00E+00

Indicator Chemical	Concentration (mg/kg)	Chronic Intake (mg/kg/day) Dermal Absorption with Incidental Ingestion via Soil
Arsenic	1.18E+01	3.39E-05
Barium*		
Benzene		0.00E+00
Chlorobenzene		0.00E+00
Chloroform		0.00E+00
Ethylbenzene		0.00E+00
Tetrachloroethene	3.21E-03	9.21E-09
Toluene		0.00E+00
1,2,4-Trichlorobenzene		0.00E+00
Trichloroethene	1.93E-03	5.54E-09
1,2,3-Trichloropropane	3.61E-02	1.04E-07
Xylenes		0.00E+00

Note: Blanks indicate none detected
*Barium is found below background levels for Eastern U.S. soils in all Off-site Operable Unit soil samples

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4.3.1 Environmental Fate and Transport

The sources of potential contamination for the Railroad Area are former discharges from the exposed bedrock wall between the Hillside Area and the railroad tracks, runoff contributed by the Seep and Hillside Areas, discharge from the Deep Aquifer, and unidentified sources connected with the operations of the railroad. Discharges of contaminants through the bedrock Table exposures were not observed during the RI field program activities, probably because of the absence of sufficient hydraulic head formerly produced by the lagoons. Discharge of ground water from the Deep Aquifer into the floodplain deposits is a potential source of contamination to the Railroad Area (ERM, 1987). However, because of the higher permeability and specific yield of the floodplain sands and gravel deposits it is believed that the quantity of ground water contributed from the bedrock aquifer to the floodplain is small in comparison to the ground water already moving through the deposits. The interpretation is supported by ground water quality data: the concentrations of organic constituents found in the shallow bedrock monitoring wells is often many orders of magnitude greater than those found in wells installed in unconsolidated floodplain deposits. This indicates that dilution within these deposits is significant. Existing residual contamination of surface and subsurface soils of the Railroad Area are therefore considered the only sources of contamination in the current assessment.

The complexity of potential transport pathways both from and within the Railroad Area is magnified by the presence of ballast material beneath the tracks. This ballast material, being of a more permeable nature than surrounding material, may act as a conduit for contaminant migration. No current estimate of the total transport of contaminants from the Railroad Area to other

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Units or movement of contaminants within the Railroad Area via the ballast can be made, however.

From the contaminated surface and subsurface soils, four modes of direct transport are possible: 1) dissolution into runoff, 2) volatilization of contaminants, 3) leaching of contaminants and discharge to the shallow flow system, and 4) generatiion of fugitive dust.

4.3.2 Potential Exposure Scenarios

The sources of contamination, the possible transport mechanisms, and the potential modes of exposure to humans from contaminants present in the Railroad Area are represented in Plate 4 (Appendix D). Based on a consideration of the source and the transport pathways, the following exposure scenarios have been identified:

1) Human exposure to contaminated runoff - Direct surface runoff in this area is probably limited because of the extremely permeable nature of the railroad bed material. Most of the runoff that occurs is collected in the drainage ditches on either side of the tracks. Collected runoff in the ditch on the southern side of the tracks is expected to reach the leachate collection system where this system is present (its estimated location is shown in Figure 1-1). Runoff that does not reach the collection system, as well as the runoff collected in the northern drainage ditch, will percolate into the surface soils or join with other sources of runoff at various culverts for combined flow in the existing drainage patterns leading to the floodplain. These existing patterns are shown in

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Figure 1-6. These drainage patterns ultimately discharge either to the swamp area in the floodplain or to the Schuylkill River. Assessment of the potential contribution of this transport mode, as well as the concentration of contaminants in surface water located on the floodplain, is addressed in the exposure assessment of the Floodplain/Wetlands Area.

2) Inhalation of Volatilized Contaminants - The low concentrations of volatile organics in the soils at the railroad tracks and the infrequency of detection suggests that the presence of volatile organics in soils in this area is extremely limited and localized? This localized presence, the absence of detection at most surface samples, and the concentrations encountered do not support evaluation of this exposure scenario.

3) Inhalation of Fugitive Dusts - While the generation of fugitive dusts from the railroad bedding material is possible, the presence of the tracks themselves and the almost continuous presence of trains prohibits reasonable quantification of dust generation. The amounts of contaminants present in this area do not indicate that inhalation of particles, if generated, would result in significant exposure. This scenario will not be evaluated further.

4) Direct Contact with Contaminated Soils - Direct human contact with contaminated soils may affect railroad workers and will be evaluated further.

5)   Human Exposure to Contaminated Ground Water - Leaching
     of contaminated soils could adversely affect ground
     water quality. Contaminants leached from the soils in
     the Railroad Area are likely to affect the shallow
     ground water flow system. The end result of any
     transport of this kind will be in the water quality of
     both surface waters on the floodplain and the
     Schuylkill River. This is currently being evaluated as
     part of both the Deep Aquifer and the
     Floodplain/Wetlands Area.

In summary, direct contact with contaminated soil particles is
considered to be an important exposure scenario.

4.3.3 Exposures to Potentially Affected Populations

Exposure of human populations to the indicator chemicals via
direct human contact with contaminated soils in the railroad area
will occur only at the Railroad Area. Soil contaminant levels
were obtained from data presented in Appendix E.

Potentially exposed populations identified at this location
include railroad workers. Exposure to this subpopulation is
expected to be covered by Occupational Safety & Health
Administration (OSHA) regulations, but is evaluated in this
document for completeness. The presence of the tracks and the
generally inaccessible nature of the Area will preclude other
groups from being exposed to the soils in this area for any
significant length of time. Although access to other Off-Site
Operable Units is gained through the Railroad Area, these
exposures would be of a very limited duration. Therefore,
transient exposures of this nature are not evaluated.

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4.3.4 Calculation of Doses and Intakes

The following route of exposure is evaluated:

1.   occupational dermal exposure to contaminated soils.

The methodology used in evaluating these exposures is presented in Section 2 of this document, and the assumptions used are presented in Table 4-5. The calculated doses and resultant intakes are presented in Table 4-6.

4.4 Seep Area

The Seep Area is bounded on the north side by the Railroad Area (Operable Unit 3), on the south side by the site access road, on the eastern edge by the Hillside Area (Operable Unit 2), and on the western edge by an unnamed tributary of the Schuylkill River. Vertically, it consists of the soils above bedrock. There are no seeps in this area. See the RI for details.

4.4.1 Environmental Fate and Transport

The sources of potential contamination for the Seep Area are non-indigenous soils used to backfill the area during a previous remedial action, as described in the Off-Site RI (ERM, 1987). No apparent hydraulic connection exists between the on-site unit and this unit (ERM, 1987).

From the source of contamination, taken to be non-indigenous contaminated surface and subsurface soils located within the unit, five modes of environmental transport are possible: 1)

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Table 4-5
Assumptions Made In the Estimation of Potential Exposure
Due To Railroad Area

Source of Exposure	Assumptions	Acceptance/Conservatism
Dermal absorption of contaminants in soil during occupational exposure of railroad workers	- Assumes that 10% of the total concentration of contaminants present in soil adhered to hands passes the dermal layer (dermal area varies with age); data on dust adherence to skin is limited, and is assumed to fall in the following range: Commercial potting soil:1.45 mg/cm2 Clay mineral kaolin:2.77 mg/cm2 - Assumes one complete coating event per day; chronic exposure scenario assumes exposure occurs 150 days per year. - Assumes exposure occurs only to adults during occupational activities	Dust adherence will depend on a variety of site specific factors; contact frequency will vary widely and will depend on a large number of factors. Approach presents probable overestimate.

Note: Assumptions for both the chronic and subchronic exposure scenarios are the same; exposure varies only as a function of the concentration of the contaminant in the media of concern.

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Table 4-5
Calculation of Potential Intakes
In The Railroad Area

Indicator Chemical	Concentration (mg/kg)	Subchronic Intake (mg/kg/day) Dermal Absorption with Incidental Ingestion via Soil
Arsenic	5.40E+00	1.37E-05
Barium*		
Benzene	7.10E-03	0.00E+00
Chlorobenzene		1.80E-08
Chloroform		0.00E+00
Ethylbenzene	2.50E-02	8.33E-08
Tetrachloroethene	1.40E-01	3.54E-07
Toluene		0.00E+00
1,2,4-Trichlorobenzene	3.80E+00	9.61E-08
Trichloroethene	5.10E-02	1.29E-07
1,2,3-Trichloropropane	1.50E-01	3.80E-07
Xylenes	1.30E-01	3.29E-07

Indicator Chemical	Concentration (mg/kg)	Chronic Intake (mg/kg/day) Dermal Absorption with Incidental Ingestion via Soil
Arsenic	4.25E+00	4.42E-06
Barium*		
Benzene		0.00E+00
Chlorobenzene	2.00E-04	2.06E-10
Chloroform		0.00E+00
Ethylbenzene	1.00E-03	1.04E-09
Tetrachloroethene	5.30E-03	5.51E-09
Toluene		0.00E+00
1,2,4-Trichlorobenzene	1.15E-01	1.20E-07
Trichloroethene	2.00E-03	2.06E-09
1,2,3-Trichloropropane	1.00E-02	1.04E-08
Xylenes	1.00E-02	1.04E-08

Note: Blanks indicate none detected
*Barium is found below background levels for Eastern U.S. soils in all Off-site Operable Unit soil samples

movement of soluble or particulate contaminants in runoff, 2) wind-induced soil erosion resulting in fugitive dust emissions, 3) volatilization of contaminants, 4) bioaccumulation, and 5) leaching and subsequent transport in the shallow flow system or bedrock aquifer.

4.4.2 Potential Exposure Scenarios

The sources of contamination, the possible transport mechanisms, and potential modes of exposure to humans are represented in Plate 5 (Appendix D). The following exposure scenarios could result from these transport pathways:

1) <u>Human exposure to contaminants in surface runoff</u> - Runoff occurs in the form of overland flow after heavy precipitation in the Seep Area. A drainage channel located along the western edge of the area appears to be the principal point of discharge from the unit. This discharge channel passes underneath the railroad tracks by way of a culvert and feeds the surface water system in the floodplain which eventually discharges into the Schuylkill River. The end result of this transport, the concentration of contaminants in the floodplain/wetland area and the Schuylkill River, is evaluated as part of the floodplain/wetland area and the Deep Aquifer exposure assessments, respectively.

2) <u>Inhalation of Fugitive Dust</u> - The presence of a perennial and dense vegetative cover on the Seep Area soils effectively reduces the probability of soil erosion. Suspension of inhalable soil particles is unlikely. This scenario will not be evaluated further.

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3) <u>Inhalation of Volatilized Contaminants</u> - The general absence of volatile organics at the Seep Area indicates that this exposure scenario is not applicable to the present conditions.

4) <u>Human Contact with Soil Contaminants</u> - The Seep Area is expected to be accessible to children of 6-12 years of age for play activities. Because of the presence of soil contamination, this exposure scenario will be evaluated further.

5) <u>Ingestion of Contaminated Soils</u> - This area is not expected to attract children exhibiting pica behavior, and as such pica behavior will not be evaluated further. However, incidental ingestion of limited amounts of soil during play activities discussed above will be evaluated.

6) <u>Bioaccumulation of Contaminants</u> - Due to the topographic gradients present in this area and the present land use, neither agricultural use by humans nor food source use by wildlife is expected. Therefore, this transport mode is not considered a viable pathway for further analysis.

7) <u>Human Exposure to Contaminants in Ground Water</u> - The potential destination for contaminants leached from Seep Area soils is either the shallow flow system or the Bedrock Aquifer. Connection to the shallow flow system is limited due to the shallow depth of soils on the southern side of the railroad tracks and the presence of the drainage ditch. However, between the

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railroad and the river there is no leachate collection system so the possibility of some connection to the shallow flow system exists. This connection is expected to be minor, however, and transport of contaminants via this mode will not be evaluated further. Discharge of contaminants to the Deep Aquifer Unit is also possible, although the overall effect of this unit on the contamination in bedrock would clearly be insignificant compared to the contribution from the DNAPL in the bedrock.

In summary, contact with contaminated soils with accompanying incidental ingestion by children 6-12 years of age will be evaluated as a potential exposure scenario resulting from contaminants present in this unit.

4.4.3 Exposure to Potentially Affected Populations

Children who reside nearby are considered to be potentially exposed populations identified at this location. The presence of the tributary may encourage recreational use by children while such use by adults is not expected.

Dermal contact with these soils will take place only at the Seep Area itself. Soil contaminant concentrations were obtained from data presented in Appendix E and are used as exposure concentrations for the dermal contact scenario.

4.4.4 Calculation of Doses and Resultant Intakes

The exposure scenario identified requires evaluation of the
following route of exposure:

1) dermal absorption of contaminants present in soils and
   accompanying incidental ingestion.

The methodology used in evaluating these exposures is presented
in Section 2 of this document, and assumptions used are presented
in Table 4-7. The calculated doses and resultant intakes from
the routes of exposure are presented in Table 4-8.

4.5 Floodplain/Wetlands Area

The Floodplain/Wetlands Area is bounded on the north side by the
Schuylkill River, on the south side by the Railroad Area
(Operable Unit 3), on the eastern and western edges by the
unnamed tributaries and consists vertically of the floodplain
sediments overlying bedrock.

4.5.1 Environmental Fate and Transport

The Floodplain/Wetlands Unit consists of a complex and
interrelated surface water/ground water system. Surface water
bodies include the intermittent eastern and western tributaries
to the Schuylkill River, other intermittent streams, the spring
originating from a concrete spring box just below the westernmost
fenced area of the On-Site Unit, a wetland area, and a series of
ponds located north of the railroad tracks (Figure 1-6). Ground

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Table 4-7

Assumptions Made In the Estimation of Exposure Incurred
Seeps Area

Source of Exposure	Assumptions	Acceptance/Conservatism
Dermal absorption of contaminants in soil during play	- Assumes that 10% of the total concentration of contaminants present in soil adhered to hands passes the dermal layer (dermal area varies with age); data on dust adherence to skin is limited, and is assumed to fall in the following range: Commercial potting soil:1.45 mg/cm2 Clay mineral kaolin:2.77 mg/cm2 - Assumes one complete coating event per day; chronic exposure scenario assumes exposure occurs 150 days per year. - Assumes exposure occurs only to children 6 to 12 years of age - Assumes incidental ingestion of 100 mg of soil in conjunction with dermal contact	Dust adherence will depend on a variety of site specific factors; contact frequency will vary widely and will depend on a large number of factors. Approach presents probable overestimate.

Note: Assumptions for both the chronic and subchronic exposure scenarios are the same; exposure varies only as a function of the concentration of the contaminant in the media of concern.

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Table 4-8
Calculation of Intakes
Seeps Area

Indicator Chemical	Concentration (mg/kg)	Subchronic Intake (mg/kg/day) Dermal Absorption with Incidental Ingestion via Soil
Arsenic	1.43E+01	1.03E-04
Barium*		
Benzene		0.00E+00
Chlorobenzene		0.00E+00
Chloroform		0.00E+00
Ethylbenzene		0.00E+00
Tetrachloroethene		0.00E+00
Toluene		0.00E+00
1,2,4-Trichlorobenzene		0.00E+00
Trichloroethene		0.00E+00
1,2,3-Trichloropropane		0.00E+00
Xylenes		0.00E+00

Indicator Chemical	Concentration (mg/kg)	Chronic Intake (mg/kg/day) Dermal Absorption with Incidental Ingestion via Soil
Arsenic	5.96E+00	1.71E-05
Barium*		
Benzene		0.00E+00
Chlorobenzene		0.00E+00
Chloroform		0.00E+00
Ethylbenzene		0.00E+00
Tetrachloroethene		0.00E+00
Toluene		0.00E+00
1,2,4-Trichlorobenzene		0.00E+00
Trichloroethene		0.00E+00
1,2,3-Trichloropropane		0.00E+00
Xylenes		0.00E+00

Note: Blanks indicate none detected
*Barium is found below background levels for Eastern U.S. soils in all Off-site Operable Unit soil samples

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water consists of the shallow flow system identified in the RI (ERM, 1987), which is recharged by the Schuylkill River, surface runoff from the south, and the Deep Aquifer Unit underlying the floodplain. Connections between the surface water and shallow-flow systems are believed to be limited. The shallow flow system probably receives a minimal amount of recharge as vertical leakage from the ponds, but, the majority of the ponded water drains to the Schuylkill River through the intermittent streams. ERM, Inc. collected discharge data during May and June 1987 from the air stripper, inflow weirs and an outflow weir to one of the wetland areas which discharges to the Schuylkill River. These data are presented in Figure 4-1 which demonstrates that there is substantial water loss from the wetland through evapotranspiration and probably seepage to ground water. During this period there was a consistent decline in the flow from all of the discharge locations accompanied by a prolonged period of zero flow conditions from the wetland to the river between May 13 and June 19.

Connection between the shallow flow system and the Deep Aquifer also appears to be limited. Data on ground water quality from wells located in the shallow flow system was collected as part of the On-Site Unit RI/FS (Baker/TSA, 1984) and indicated relatively low concentrations of contaminants in this system (ERM, 1987). Recharge to the shallow flow system from the Deep Aquifer may occur but its effect appears to be minimized by dilution in the shallow flow system. The spring is also a potential connection between the two systems, although its role is unclear. Its origin appears to be in bedrock, but the spring box depth is unknown at this time. Residual contaminants are adsorbed to soils and sediments and are potentially present as dissolved constituents in the surface waters. The undefined nature of

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TYSON'S FLOODPLAIN

FLOW RATES

+   Discharge to the Schuylkill River
    from wetland area

□   Air stripper discharge

◇   Influent discharge to wetland area
    (includes air stripper discharge)

FLOW (gpm)
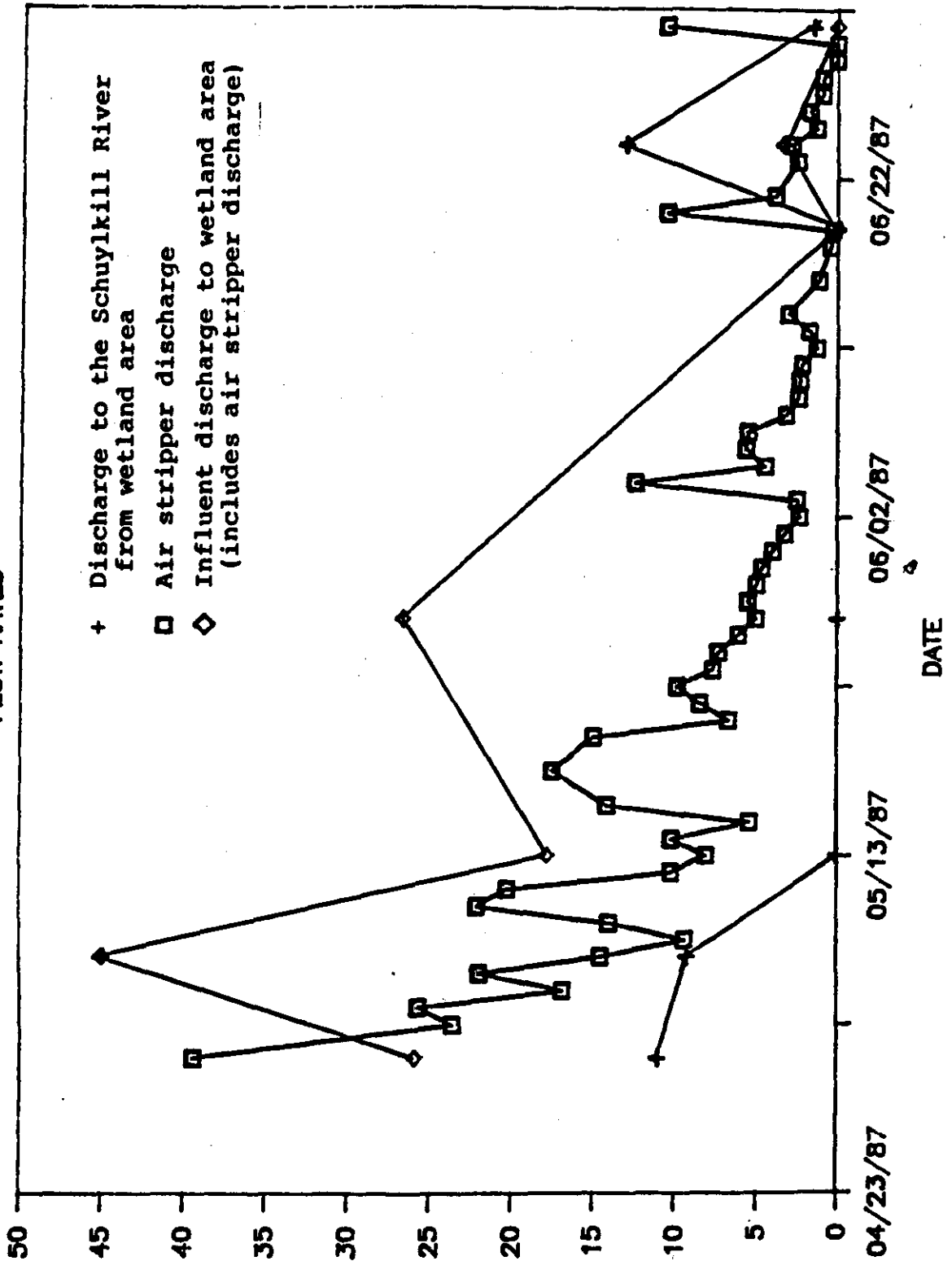
DATE

surface water/ground water relationships does not allow
identification of the primary sources of contamination for this
Unit. Therefore, surface water bodies and the associated
sediments of the Floodplain/Wetlands Area will be considered as
the sources of contamination for the purposes of this EA.

From the sources of contamination, taken to be seasonal surface
water located on the floodplain and the associated sediments,
three modes of transport are possible: 1) volatilization of
contaminants, 2) discharge to perennial surface water units, and
3) bioaccumulation.

4.5.2 Potential Exposure Scenarios

The potential sources of contamination, possible modes of
transport, and potential routes of human exposure for the
floodplain area are presented in Plate 6 (Appendix D). The
following exposure scenarios are possible from these modes of
transport:

1) <u>Ingestion of contaminated plants and biota, including
   fish</u> - Samples of wetland plants and river fauna,
   including a snapping turtle and Asiatic clams, were
   collected to determine the occurrence of
   bioconcentration of compounds or constituents.
   Analyses for the samples included Hazardous Substance
   List (HSL) volatiles, semi-volatiles (both base
   neutrals and acid extractables), pesticides/PCBs, and
   1,2,3-trichloropropane. Although a detailed pathologic
   examination could not be conducted on the frozen turtle
   specimens, the turtles were inspected for gross
   abnormalities. Neither turtle showed evidence of gross
   abnormalities.

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No detectable concentrations of 1,2,3-trichloropropane, a predominant site contaminant and a major component of the dense nonaqueous phase liquid present in the deep aquifer, were reported in any of the biological samples. The detection limit for the 1,2,3-trichloropropane was reported to be 5 ppb for these samples. However, as stated in the ERM data review (ERM, 1987), the extended period of sample storage prior to analysis may have resulted in losses of VOA analytes including 1,2,3-trichloropropane from the sample.

Because of excessive sample holding time by the laboratory, the reported results for volatile organic compounds (VOCs) cannot be regarded with any measure of confidence. The VOCs reportedly detected in tissue samples are common laboratory solvents and can easily adulterate samples stored for long periods. It may be noted in this context that no detections of trichloropropane, which is not a common laboratory contaminant, were reported in these samples.

According to the Superfund Public Health Evaluation Manual (1986), the bioconcentration factors for VOCs are relatively low (those for benzene and chloroform are 5.2 and 3.75, respectively). Biomagnification of these compounds is of minimal significance.

In summary, bioaccumulation of the compounds of concern has not been demonstrated to occur, and ingestion of aquatic species by humans or wildlife is not expected to be a significant pathway of exposure. It is not evaluated further. In addition, harvest of terrestrial

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resources by hunting is restricted by township ordinance forbidding the discharge of firearms, and trapping of muskrats is carried out for fur collection, not food collection. Exposure via ingestion of terrestrial species is not evaluated further.

2)   Inhalation of volatilized compounds - Inhalation of volatilized contaminants from Floodplain sediments will be considered. Evaluation of volatilization of contaminants from surface water bodies will also be evaluated.

3)   Human exposure to surface water - The Floodplain/Wetland Area is expected to be accessible to children. Exposures through recreational use of water will be evaluated for children 6-12 years of age only; children of a younger age are not expected to be available for extended periods of unsupervised play.

4)   Human exposure to contaminated soils and sediments - Contaminated sediment is the primary affected medium in this unit and exposure associated with play activities of children 6-12 years of age will be evaluated further. Some contaminated sediment can also be expected to be transported during episodic flooding events. Sediment transported in this manner is expected to be retained in the riverine system, and is of minor significance related to the potential quantities of contaminated sediment resulting from the direct discharge of contaminated ground water to the river.

5)   Ingestion of contaminated soils - Exhibition of pica behavior was not modelled due to 1) children of pica

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age (2-6 years) are not expected to cross the large number of railroad tracks necessary to reach the Floodplain/Wetland Area, and 2) such exposure, if it did occur, is expected to be an isolated incident and not appropriate for evaluation as a chronic exposure. Incidental ingestion of soils during play activities of children 6-12 years of age will be evaluated in conjunction with dermal exposure to contaminated soils.

6)   Human exposure to off-site surface water bodies - Surface water discharging from the Floodplain/Wetlands Area to the Schuylkill River is a minor potential source of contamination to this surface water body relative to the discharge of contaminated ground water from the bedrock aquifer (ERM, 1987). The assessment of exposures attributed to this ultimate discharge point, the Schuylkill River, is therefore carried out as part of the assessment of the Deep Aquifer.

In summary, dermal contact with contaminants present in sediments or surface water by children 6-12 years of age and inhalation of contaminants volatilized from soils and surface water will be evaluated as potential modes of exposure.

4.5.3 Exposures to Potentially Affected Populations

Consideration of the modes of transport identified for further analysis indicates that exposure of human populations to the indicator chemicals will occur only at the floodplain itself. The amount of contaminant adsorbed onto floodplain sediments and data on water quality were obtained from results presented in Appendix E. The overriding source of exposure in this area is dermal contact with contaminated soils; inhalation contributes

less than 1% of the total intake of indicator compounds, and will therefore not be carried further in the analysis.

Adults and children who use the floodplain and river area for recreation are potentially exposed populations.

4.5.4 Calculation of Doses and Resultant Intakes

Adsorption of contaminants onto floodplain sediments indicates evaluation of the following routes of exposure for the potentially exposed populations:

1) dermal absorption of contaminants, and

2) ingestion of contaminants.

The methodology used for calculation of doses and intakes is presented in Section 2. Assumptions used in these calculations are presented in Table 4-9. The doses and resultant intakes from these routes of exposure are presented in Table 4-10.

4.5.5 Environmental Assessment

An extensive field investigation was carried out in the floodplain/wetland area to identify plant and animal species present, characterize any impairment of wetland function if present, analyze the potential mobility of organic chemicals in this area, investigate potential toxicity of contaminants present, and determine the potential of these contaminants to

Table 4-9
Assumptions Made in the Estimation of Exposure Incurred
Floodplain/Wetland Area

Source of Exposure	Assumptions	Acceptance/Conservatism
Dermal absorption of contaminants in soil during play	- Assumes that 10% of the total concentration of contaminants present in soil adhered to hands passes the dermal layer (dermal area varies with age); data on dust adherence to skin is limited, and is assumed to fall in the following range: Commercial potting soil:1.45 mg/cm2 Clay mineral kaolin:2.77 mg/cm2 - Assumes one complete coating event per day; chronic exposure scenario assumes exposure occurs 150 days per year. - Assumes exposure occurs only to children 6 to 12 years of age - Assumes incidental ingestion of 100 mg of soil in conjunction with dermal contact	Dust adherence will depend on a variety of site specific factors; contact frequency will vary widely and will depend on a large number of factors. Approach presents probable overestimate; limited behavioral information available.
Dermal absorption of contaminants in surface water during play	- Assumption is that VOCs pass the dermal layer in proportion to the flux of water, and that 100% is absorbed. - Assumes 30 minutes of exposure to 20% of total body surface area; chronic exposure scenario assumes exposure occurs 150 days per year - Assumes exposure occurs only to children 6 to 12 years of age	Reality of absorption phenomena is unknown; length and extent of exposure presents probable overestimate; limited behavioral information available.

Note: Assumptions for both the chronic and subchronic exposure scenarios are the same; exposure varies only as a function of the concentration of the contaminant in the media of concern.

Table 4-10
Calculation of Intakes
Floodplain/Wetland Area

Indicator Chemical	Concentration in S. Water (mg/l)	Concentration in Soil (mg/kg)	Concentration in Air (mg/m3)	Children 2-6 years Subchronic Intakes (mg/kg/day) Ingestion of Sediments	Inhalation via Soils and S.Water	Children 6-12 years Subchronic Intakes (mg/kg/day) Dermal Absorption via Soil and S. Water	Inhalation via Soils and S.Water
Arsenic Barium*	1.10E-02	2.60E-01		0.00E+00	0.00E+00	1.90E-04	0.00E+00
Benzene	7.00E-03	2.00E-01		0.00E+00	0.00E+00	9.00E-06	0.00E+00
Chlorobenzene		1.10E-02		0.00E+00	0.00E+00	2.01E-06	0.00E+00
Chloroform	2.00E-03	5.70E-01		0.00E+00	0.00E+00	7.96E-08	0.00E+00
Ethylbenzene	1.20E-02	5.00E-02		0.00E+00	0.00E+00	4.19E-06	0.00E+00
Tetrachloroethene	6.00E-02	2.20E-01		0.00E+00	0.00E+00	5.76E-07	0.00E+00
Toluene	2.60E-02	3.20E-01		0.00E+00	0.00E+00	2.82E-06	0.00E+00
1,2,4-Trichlorobenzene	1.00E-02	4.00E-02		0.00E+00	0.00E+00	2.36E-05	0.00E+00
Trichloroethene	1.70E+00	6.30E+00		0.00E+00	0.00E+00	5.76E-07	0.00E+00
1,2,3-Trichloropropane	1.90E-01	1.60E+00		0.00E+00	0.00E+00	7.62E-05	0.00E+00
Xylene				0.00E+00	0.00E+00	1.50E-05	0.00E+00

Indicator Chemical	Concentration in S. Water (mg/l)	Concentration in Soil (mg/kg)	Concentration in Air (mg/m3)	Children 2-6 years Chronic Intakes (mg/kg/day) Ingestion of Sediments	Inhalation via Soils and S.Water	Children 6-12 years Chronic Intakes (mg/kg/day) Dermal Absorp. via Soils and S.Water	Inhalation via Soils and S.Water
Arsenic Barium*	3.00E-03	1.05E-01		0.00E+00	0.00E+00	3.92E-05	0.00E+00
Benzene	3.04E-04	2.70E-02		0.00E+00	0.00E+00	0.00E+00	0.00E+00
Chlorobenzene		7.66E-04		0.00E+00	0.00E+00	7.97E-08	0.00E+00
Chloroform	4.17E-05	4.25E-02		0.00E+00	0.00E+00	2.25E-08	0.00E+00
Ethylbenzene	2.90E-04	5.67E-03		0.00E+00	0.00E+00	1.29E-07	0.00E+00
Tetrachloroethene	1.30E-03	2.92E-02		0.00E+00	0.00E+00	1.79E-08	0.00E+00
Toluene	4.90E-04	3.01E-01		0.00E+00	0.00E+00	9.32E-08	0.00E+00
1,2,4-Trichlorobenzene	3.60E-04	3.13E-03		0.00E+00	0.00E+00	6.67E-07	0.00E+00
Trichloroethene	1.39E-01	4.65E-01		0.00E+00	0.00E+00	1.11E-08	0.00E+00
1,2,3-Trichloropropane	3.60E-03	1.90E-01		0.00E+00	0.00E+00	2.34E-06	0.00E+00
Xylene				0.00E+00	0.00E+00	5.43E-07	0.00E+00

Note: Blanks indicate none detected
*Barium is found below background levels by Eastern U.S. soils in all Off-site Operable Unit soil samples

bioaccumulate in species present in the area. The results of
this investigation, as reported in detail in the RI, are
presented in Appendix F for reference.

The floodplain/wetland area appears to support a diverse and
unimpacted flora and associated fauna. No visible impairment of
wetland functions has occurred. Bioaccumulation studies reported
no verified bioaccumulation of any site related compound.
Sediment toxicity bioassays performed as additional requested
work showed no effects due to site-related compounds.

SECTION 5

TOXICITY EVALUATION

5.1 Evaluation Process

The toxicity evaluation of the selected indicator chemicals for
the Tyson's site follows the procedure outlined in Section 2.4.
The process involves three components:

- the designation of carcinogenicity
- the evaluation of noncarcinogenic risk
- the comparison with environmental standards

The decision to classify a compound as a potential carcinogen has
serious consequences for the conduct of quantitative risk
assessments. Wrongly attributing a compound's carcinogenicity
can result in severe over- or under-estimations of carcinogenic
risk. As carcinogenic risk at CERCLA sites is normally the most
restrictive component of the Endangerment Assessment process, the
appropriateness of cleanup decisions quite likely depends upon
the accuracy of the determination of carcinogenic risks.

Noncarcinogenic risks are evaluated primarily by comparing
site-related doses to acceptable daily intakes established to
protect against various types of acute and chronic effects.

Comparisons with environmental standards are also essential to
the understanding of site-related levels of environmental risk.

A summary of toxicological information for the indicator chemicals is presented in Table 5-1, which includes the environmental standards, acceptable daily intakes for noncarcinogenic effects and potency factors for potential carcinogens.

5.2 General Principles of Toxicology

Exposure to a toxic chemical pollutant does not necessarily result in adverse effects. The relationship between dose and response defines the quantitative indices of toxicity required to evaluate the potential health risks associated with a given level of exposure.

If the nature of the dose-response relationship is such that no effects are observed or can be assumed to occur below a certain level of exposure, a threshold can be defined and an acceptable exposure level derived. Humans are routinely exposed to naturally occurring and man-made chemicals at low levels through the typical diet, through air and water. As the level of exposure exceeds a threshold, there exists an probability that some of the most sensitive members of the population will show adverse effects.

Adverse effects are considered to be functional impairments or pathological injuries that could affect the biological integrity of the living organism, or that reduce an organism's ability to effectively respond to an additional insult.

Adverse effects can be caused by acute exposure, which is a single or short-term exposure to a toxic substance, or by chronic exposure to lower levels on a continuous or repeated basis over

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TABLE 6-1

SUMMARY OF TOXICOLOGICAL INFORMATION
FOR THE INDICATOR CHEMICALS

(CONCENTRATIONS IN mg/L UNLESS OTHERWISE SPECIFIED)

KEY:
PR = PRIMARY DRINKING WATER STANDARD
HBN = HEALTH BASED NUMBER EPA OFFICE OF SOLID WASTE
I = 51 FR 1949
N.A. = NOT APPLICABLE
BB = EPA 1972 WATER QUALITY CRITERIA (BLUE BOOK)
EDE = EQUIVALENT DOSE ESTIMATE FROM OFFICE OF SOLID WASTE

* For the purposes of the Endangerment Assessment, ERM has made the assumption that TCP is 0.02 carcinogen and developed an innate potency factor of 1.98 E-01 ((mg/kg/day)) as described in Appendix H.

an extended period of time. "Acceptable" acute or chronic levels of exposure are considered to be without any anticipated adverse effects. Such exposure levels are commonly expressed, derived, or represented in terms such as ADI (Acceptable Daily Intake), MCL (Maximum Contaminant Level), NOAEL (No Observable Adverse Effect Level), AIC (Acceptable Chronic Intake), AIS (Acceptable Subchronic Intake), SNARL (Suggested No-Adverse-Response Level), AWQC (Ambient Water Quality Criteria), TLV (Threshold Limit Value), etc. An acceptable exposure level is calculated to provide an "adequate margin of safety."

Most chemical substances exhibit threshold biological effects. There is debate in the scientific community over whether thresholds exist for carcinogens. However, cancer-producing agents have generally been assumed by EPA not to exhibit thresholds. This is not to say that thresholds may not exist for some or all carcinogens. However, as stated in the 1977 report of the National Academy of Science's Safe Drinking Water Committee: "The idea that there is a safe dose of such chemicals may be conceptually valid, but safety cannot be established by any experimental method now available." It has, therefore, been assumed by EPA that any exposure would represent some finite level of risk. Depending upon the potency of the carcinogenic agent and the level of exposure, such a risk could be vanishingly small at very low doses.

For carcinogenic substances, accordingly, acceptable chronic exposure levels (ADIs) are generally not derived. Rather, probabilities that a specific adverse effect (viz., tumor growth) will occur are derived using mathematical models, generally applied to experimental data on animals exposed to high dose levels. These probabilities are then extrapolated to estimate the quantitative risk posed to humans exposed to vastly lower

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concentration in the environment. This numerical estimate of risk is commonly expressed as UCR (Unit Cancer Risk), which is defined as the excess or added risk of cancer due to a continuous lifetime exposure to one unit of carcinogen (e.g., cancer risk at 1 one-thousandth of a gram of carcinogenic chemical per kilogram body weight per day for a lifetime). Such estimates are designed to be highly conservative (i.e., the true risk is most probably much lower than the estimate). The linear extrapolation model recommended by EPA provides an estimate of the upper limit of risk that involves numerous, unverifiable assumptions, and a high degree of uncertainty.

The term "carcinogen" is often misapplied to differentiate a list of "dangerous" compounds from those that are "safe." However, there is a wide range in potencies among animal tumorigens with widely varying degrees of carcinogenic evidence. EPA recently proposed an approach for classifying chemicals based upon the strength of evidence of carcinogenicity. The categorization consists of a five-category approach, as follows:

Group A-   Human carcinogen (sufficient evidence from epidemiological studies)

Group B-   Probable human carcinogen

   Group B1-   At least limited evidence of carcinogenicity to humans

   Group B2-   Usually a combination of sufficient evidence in animals and inadequate data in humans

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Group C-   Possible human carcinogen (limited evidence of
                carcinogenicity in animals in the absence of human
                data)

Group D-   Not classified (inadequate evidence of animal
                carcinogenic activity)

Group E-   No evidence of carcinogenicity for humans (no
                evidence for carcinogenicity in at least two
                adequate animal tests in different species or in
                both epidemiological and animal studies).

5.3 Level of Evidence for Carcinogenicity

The level of evidence for carcinogenicity for the indicator
compounds is discussed in detail in Appendix G. A brief summary
of that discussion is given below.

The following compounds have been found in appreciable
concentrations off the Tyson's site: arsenic,
1,2,3-trichloropropane, chloroform, benzene, and
tetrachloroethylene, and trichloroethylene.

Both EPA and IARC have classified arsenic, chloroform, and
benzene as human carcinogens. EPA's carcinogenic potency factor
for arsenic by inhalation is 2×10^1 $(mg/kg/day)^{-1}$ which places
it among the most potent of the potential carcinogens evaluated
to date by EPA's Carcinogen Assessment Group (CAG). The potency
factor for arsenic is currently being re-evaluated and the
potency factor for arsenic has been debated during the
Environmental Risk Forum. The carcinogenic potency factor for

arsenic may be lowered at least an order of magnitude in the future. Arsenic will be treated as a noncarcinogen for this assessment since no inhalation exposure scenarios are examined. Ingestion scenarios are included, but arsenic is considered not to be carcinogenic by oral exposure. This is because, in establishing drinking water standards, EPA has treated arsenic as if it were not carcinogenic by ingestion. EPA's carcinogenic potency factor for benzene is 4.45×10^{-2} $(mg/kg/day)^{-1}$ which places it among the least potent of the potential carcinogens evaluated to date by CAG. EPA's carcinogenic potency factor for chloroform is 8.1×10^{-2} $(mg/kg/day)^{-1}$ which places it among the least potent of the potential carcinogens evaluated to date by CAG.

There is a significant controversy in the international scientific community surrounding the classification of trichloroethylene (TCE) and tetrachloroethylene (PCE). EPA has classified them as probable (Class B2) carcinogens. This classification was based on observations of mouse liver tumors, the significance of which is the subject of extreme controversy in the scientific community. For example, IARC considers mouse liver tumor alone as insufficient evidence of carcinogenicity. Similar considerations have been discussed by the Office of Science and Technology Policy, the Nutrition Foundation and the National Toxicology Program. EPA's interpretation of mouse liver tumors observed in long-term studies and the use of the linearized multistage model for calculation of carcinogenic potency have not been widely accepted by the scientific community. IARC has determined that there is insufficient evidence to classify TCE and PCE as regards carcinogenicity at this time. In the present Endangerment Assessment for the Tyson's Off-Site Remedial Investigation, ERM is treating TCE and PCE by EPA's procedures and has included them in the carcinogenic risk

assessment. However, the classification of TCE and PCE inevitably increases the potential for over-estimating carcinogenic risks.

On the basis of a communication in early 1987 between EPA Region 3 toxicologists and the National Toxicology Program (NTP), regional EPA has adopted the position that TCP should be regarded as a probable human carcinogen (Appendix G). NTP informed Region 3 that an "alert" notice concerning the preliminary, mid-course findings of carcinogenic activity of TCP in rats and mice was to be issued by early May, 1987. (No "alert" has been released by NTP as of this writing, July 28, 1987).

TCP has not at this time been evaluated by EPA's Carcinogen Assessment Group. The NTP is currently conducting the pathology evaluation stage of the bioassay which is expected to reach completion by the end of the year. Following internal and external review, the conclusions of the study regarding the possible carcinogenicity of TCP in animals are anticipated to be released sometime in early 1988.

For purposes of Endangerment Assessment, it is assumed that the animal bioassay results will indicate a B2 classification for TCP. ERM has developed an interim potency index based on the geometric mean of the EPA potency factors of five structurally related chlorinated hydrocarbons as described in detail in Appendix G. The interim potency factor of 1.0×10^{-1} (mg/kg/day)$^{-1}$ will be used in the risk assessment process for purposes of risk characterization under an assumption of cancer-causing potential of TCP in humans.

It is important to emphasize that details concerning the NTP mid-course study results are unavailable for any independent

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assessment and review to determine the significance of the data.
The results of a properly reviewed completed study may change the
interim status of TCP and result in different potency factors.

Both EPA and IARC consider that toluene, xylene,
1,2,4-trichlorobenzene, chlorobenzene, ethylbenzene, and barium
display no evidence of human carcinogenicity. A fuller
discussion of the noncarcinogenic and carcinogenic effects of the
indicator chemicals is provided in Appendix H.

5.4 Comparison with Environmental Standards

As discussed in Section 5.1, evaluation of exposure point
concentrations compared with environmental standards is an
important part of the CERCLA Endangerment Assessment process.
Applicable or relevant and appropriate requirements for each
indicator chemical are presented in Table 5-1.

SECTION

6

SECTION 6

RISK CHARACTERIZATION

This section assesses the potential risks to human health and the
environment associated with exposure to the various indicator
chemicals under existing conditions. The risks of exposure to
carcinogens and noncarcinogens are assessed separately by
comparing:

1. current exposure point concentrations with applicable
    or relevant and appropriate standards,

2. current acute doses with acceptable subchronic intakes
    for carcinogens and noncarcinogens,

3. current chronic doses of noncarcinogens with acceptable
    chronic intakes, and

4. calculated risks with acceptable risks for potential
    carcinogens.

A discussion of uncertainties encountered in the risk assessment
process is included in this section to provide some perspective
in interpreting the results of the assessment.

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6.1 Comparison to Applicable and Relevant Standards

Identification of possible ARARs depends upon the recognized uses and designations of the resources of concern. ARARs for the Off-Site Operable Units were divided into media categories. As applicable or relevant standards do not exist for concentrations of these compounds in soil, comparison of the concentrations of contaminants in soil at any of the Operable Units with standards is not possible. Two subclasses of ARARs exist under each category; those legally applicable (i.e., enforceable) to the substance and those that might be deemed relevant and appropriate under the circumstances of the release.

The resources of media of concern, as identified in the exposure pathways analysis, included air, ground water and surface water (Schuylkill River). The classification and use of each resource and the potential ARARs are presented as follows:

- Air

    Montgomery County, Pennsylvania, where the Tyson's Site is located, is in a non-attainment area for ozone as per the National Ambient Air Quality Standards (NAAQS) promulgated under the Clean Air Act. The NAAQS are enforceable standards applicable at acceptable ambient air monitoring locations. 25 PA Code §127.11 requires a plan approval for air strippers and other equipment designed to remove volatile contaminants from soil, water, and other materials. Ambient Air Quality Guidelines listed within the Interim Operating Guidelines for Air Toxic Substances (ATGs) are possible ARARs; exemptions may be granted from the permit requirements to minor significance provisions if: (1)

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stack concentrations of each individual air toxic constituent does not exceed one-third of the ATG ambient guideline concentrations, and (2) potential (before control) emission rates of all listed air toxics do not exceed a total of one pound per hour.

- Ground Water

Ground water beneath the Tyson's Site, between the former lagoon area and the river, is not presently used for drinking water, household, or other use. The exposure point of concern related to ground water contaminants, assuming that some discharge of ground water to the river occurs, would be the Schuylkill River.

- Surface Water (Schuylkill River)

Protected uses designated for the Schuylkill River are: propagation of warm water fish, water supply for all purposes (industrial, domestic, agricultural, wildlife), fishing, water contact sports, natural areas, power generation, and assimilation of treated wastes. Uses not now protected on the Schuylkill River include: cold water fish, migratory fish, boating, conservation, and navigation.

Federal Ambient Water Quality Criteria (WQC) for the protection of human health are ambient concentration guidelines, and are potential ARARs. Federal WQC for the protection of aquatic life and Pennsylvania Water Quality Criteria are possible ARARs and are applied at the point of discharge.

Safe Drinking Water Act Maximum Contaminant Levels
(MCLs) are possible ARARs applied "at the tap", or at
the point of human consumption.

Potential applicable and relevant standards, as defined in the
Draft Superfund Public Health Evaluation Manual, for all
compounds detected within the Deep Aquifer and the Floodplain/
Wetlands Area are listed in Table 6-1.

6.2 Calculation of Subchronic Hazard

Total subchronic hazard for each potentially exposed population
for each Operable Unit is presented in Table 6-2. The hazard
index is the ratio of the expected potential dose and acceptable
exposure levels. Values of less than one indicate that no
subchronic hazard exists.

6.3 Calculation of Noncarcinogenic Hazard

The assessment of noncarcinogenic hazard for for the Deep
Aquifer, the Hillside Area, the Railroad Area, the
Floodplain/Wetlands Area, and the Seep Area are shown in Table
6-3. The hazard index is the ratio of the expected potential
dose and acceptable exposure levels. Values of less than one
indicate that no noncarcinogenic hazard exists. The exposure to
noncarcinogenic risk is solely via dermal contact with or
ingestion of contaminated soils. Even if all effects are assumed
to be additive, it is apparent from Table 6-3 that no significant
noncarcinogenic hazard exists.

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TABLE 6-1
COMPARISON TO POTENTIAL APPLICABLE OR RELEVANT AND APPROPRIATE REQUIREMENTS
(Concentrations in mg/L)

Area/Unit	Compound	Mean	Maximum (mg/L)	Safe Drinking Water Act MCL	Final MCLG	Proposed MCLG	Freshwater Aquatic Life Acute Toxicity Criteria	Freshwater Aquatic Life Chronic Toxicity Criteria	Human Health Criteria Water and Fish Consumption	Existing Pennsylvania Water Quality Standards
DEEP AQUIFER (Schuylkill River)	1,2,3-Trichloropropane	2.23E-04	3.50E-04							
	Xylenes	3.53E-04	7.00E-03			4.40E-01				
FLOODPLAIN/WETLAND Surface Water	Aluminum	3.23E-01	6.90E+00							
	Chromium	4.00E-03	1.00E-02	5.00E-02		1.20E-01	1.70E+00	2.10E-01	1.70E+02	5.00E-02
	Barium	1.15E-01	4.00E-01			1.50E+00			1.00E+00	
	Copper	1.70E-02	5.00E-02			1.30E+00	1.80E-02	1.20E-02	3.00E-01	1.00E-01
	Iron	1.52E+00	1.64E+01					1.00E+00	5.00E-02	1.50E+00
	Manganese	8.89E-01	3.75E+00							1.00E+00
	Arsenic	3.00E-03	1.10E-02	5.00E-02		5.00E-02	3.60E-01	1.90E+00		5.00E-02
	Zinc	3.50E-02	9.00E-02				3.20E-01	4.70E-02		
	Lead	2.00E-03	8.30E-03	5.00E-02		2.00E-02	8.20E-02	3.20E-03	5.00E-02	
	Acetone	4.33E-03	1.30E-01							
	Methylene chloride	1.51E-03	7.50E-03							
	Toluene	3.05E-03	6.80E-02			2.00E+00	1.75E+01		1.43E+01	
	Total xylenes	9.10E-03	1.90E-01			4.40E-01				
	2-Butanone	0.00E+00	0.00E+00							
	Chlorobenzene	3.04E-04	7.00E-03						4.00E-01	
	Chloroform	3.41E-04	7.50E-03				2.89E+01	1.24E+00	1.90E-04	
	trans-1,2-Dichloroethene	1.56E-04	7.50E-03							
	Trichloroethene	1.44E-03	1.60E-02							
	4-Methyl-2-pentanone	1.96E-04	7.50E-03							
	1,2,3-Trichloropropane	1.39E-01	1.70E+00							
	Ethylbenzene	4.17E-05	2.00E-03			6.80E-01	3.20E+01		1.40E+00	
	Carbon disulfide	1.13E-04	5.40E-03							
	1,2-Dichlorobenzene	1.14E-03	3.30E-02			6.20E-01	1.11E+00	7.63E-01	4.00E-01	
	1,4-Dichlorobenzene	3.48E-04	1.00E-02	7.50E-01	7.50E-01		1.11E+00	7.63E-01	4.00E-01	
	Phenol	1.74E-04	8.00E-03				1.02E+01	2.56E+00	3.50E+00	
	4-Methylphenol	2.17E-03	1.00E-01							
	Butyl benzyl phthalate	0.00E+00	0.00E+00							
	Bis (2-ethylhexyl) phthalate	0.00E+00	0.00E+00						1.50E+01	
	DDE	1.09E-06	5.00E-05							
	DDD	1.09E-05	2.00E-04				1.05E+00			
	PCB-1248	7.61E-05	3.50E-03			0.00E+00	2.00E-03	1.40E-05	7.90E-06	

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Table 3-2
Calculation of Noncarcinogenic Hazard Indices

Chemicals (column rows, repeated per receptor group):
- Arsenic
- Barium
- Chloroform
- Chlordane
- Ethylbenzene
- Toluene
- 1,2,4-Trichlorobenzene
- Trichloroethene
- 1,2,3-Trichloropropane
- Xylene

Receptor groups:
- Deep Aquifer / Adult
- Middle Aquifer Children 6-12
- Bedrock Aquifer Adult
- Deep Aquifer Children 6-12

6.4 Calculation of Carcinogenic Risk

Assessment of potential carcinogenic risks for existing site conditions within the Hillside Area, the Railroad Area, the Floodplain/Wetlands Area and the Seep Area are presented in Table 6-4 and are in the range which is considered acceptable by EPA (less than 10^{-6}).

Proper assessment of potential carcinogenic risk for existing conditions in the Deep Aquifer, i.e., assessment of potential carcinogenic risk posed by household and/or drinking water use of contaminated surface water, requires assessment of both the risk posed by background levels of contaminants present in the water supply as well as the additional risk posed by the presence of site-related compounds. Both sources of risk can be estimated using data generated by water distribution companies and from data generated as part of the RI (ERM, 1987). Since 1979, PSWA has been collecting samples of the raw and treated water at both Philadelphia intakes and analyzing for various organic compounds. These analyses were performed by gas chromatography (GC) techniques with detection limits as low as 100 parts per trillion (ppt). However, the data prior to June 1986 should be used cautiously since confirmatory techniques (i.e., GC-Mass Spectroscopy) were not historically performed. In fact, prior to July 1982 less sensitive analytical techniques were employed and these earlier data are of limited value in attempting to ascertain any historical pattern of organic compounds in the low ppt range.

Review of the data from the PSWA Philadelphia intakes shows that 1,2,3-trichloropropane (TCP) is a consistently detected compound in the raw Schuylkill River water. The most often detected

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Table 9-4
Estimation of Carcinogenic Risk

compounds in the treated water at both water treatment plants are TCP and the trihalomethanes. All available information obtained to date indicate that the probable source of the TCP is ground water discharging from the bedrock aquifer to the Schuylkill River at the Tyson's Superfund Site. The trihalomethanes are most probably a result of chlorination used at the two Philadelphia water treatment plants. Table 6-5 shows the average annual concentrations of TCP and trihalomethanes in both the raw and treated water at the Queen Lane and Belmont Treatment Plants. Only data since 1982 has been averaged and included on this Table because of the implementation of more sophisticated analytical techniques during this time period.

In May 1986, EPA Region III confirmed the presence of TCP in the raw water at the intakes of all three water treatment plants on the Schuylkill River downriver of the Tyson's Site. The presence of TCP in both the raw water and treated water at all three of the treatment plants was confirmed through samples obtained during the Off-Site Operable Unit Remedial Investigation at the Tyson's Site. ERM data reported in the RI (ERM, 1987) showed similar concentrations of TCP in the Belmont and Queen Lane Treatment Plant Water samples.

Average effluent concentrations calculated over the time period 1982-1985 for both the Belmont and Queen Lane Treatment Plants were used as input for estimation of calcinogenic risk posed by both background levels of organic compounds and site-related contaminants. This time period was selected on the basis of availability of data for all compounds. The estimation of carcinogenic risk from these levels of compounds is presented in Table 6-6 and Figure 6-1. For Belmont Treatment Plant effluent, trihalomethanes contribute 85% of the carcinogenic risk estimated

TABLE 6-5

ANNUAL AVERAGE CONCENTRATIONS OF 1,2,3-TRICHLOROPROPANE AND TRIHALOMETHANES IN THE INFLUENT AND EFFLUENT WATER AT THE PHILADELPHIA WATER DEPARTMENT INTAKES.

(concentrations in ug/L)

	1,2,3-TRICHLOROPROPANE		TRIHALOMETHANES	
	Queen Lane Influent	Belmont Influent	Queen Lane Influent	Belmont Influent
1982-1983	0.17	0.33	0.38	0.27
1983-1984	0.13	0.17	0.23	0.18
1984-1985	0.18	0.18	0.18	0.19
1985-1986	0.06	0.05	0.14	0.13
1986-1987	0.22	0.21	NA	NA
Average	0.15	0.19	0.23	0.19
ERM Data	0.17	0.16		

	Queen Lane Effluent	Belmont Effluent	Queen Lane Effluent	Belmont Effluent
1982-1983	0.18	0.36	51.83	46.33
1983-1984	0.13	0.24	45.48	48.48
1984-1985	0.16	0.21	46.48	52.75
1985-1986	0.09	0.08	47.04	46.78
1986-1987	0.19	0.26	NA	NA
Average	0.15	0.23	47.71	49.09
ERM Data	0.19	0.13		

Table 6-3
Estimates of Carcinogenic Risk from Contaminants Found in Treatment Plant Effluent

Distant Treatment Plant Effluent

Chemical	Concentration (mg/l)	Ingestion of Drinking Water	Dermal Absorption in Showering/Bathing	Inhalation via Showering/Bathing	Inhalation of Household Air	Carcinogenic Potency Factor (1/mg/kg/day) Oral	Inhalation	Carcinogenic Risk (Noncarcinogen)
Trichloroethene (PCE)								
1,2,3-Trichloropropane								
Other Carcinogens:								
Methylene Chloride								
1,2-Dichloroethane								
Carbon Tetrachloride								
1,1,2-Trichloroethane								
Bis(2-chloroethyl)ether								
Tetrachloroethene								
Benzene								

Close Line Treatment Plant Effluent

Chemical	Concentration (mg/l)	Ingestion of Drinking Water	Dermal Absorption in Showering/Bathing	Inhalation via Showering/Bathing	Inhalation of Household Air	Carcinogenic Potency Factor (1/mg/kg/day) Oral	Inhalation	Carcinogenic Risk (Noncarcinogen)
Trichloroethene (PCE)								
1,2,3-Trichloropropane								
Other Carcinogens:								
Methylene Chloride								
1,2-Dichloroethane								
Carbon Tetrachloride								
1,1,2-Trichloroethane								
Bis(2-chloroethyl)ether								
Tetrachloroethene								
Benzene								

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Carcinogenic Risk Posed by Drinking Water Constituents

Figure axes: Y-axis labeled "Excess Cancer Deaths per Million Exposed" (0 to 600). Bars for Belmont and Queen's Lane.

Belmont:
- Trihalomethanes (total)
- 1,2,3-Trichloropropane (3)
- Other Carcinogens (total)

Queen's Lane:
- Trihalomethanes (total)
- 1,2,3-Trichloropropane (2)
- Other Carcinogens (total)
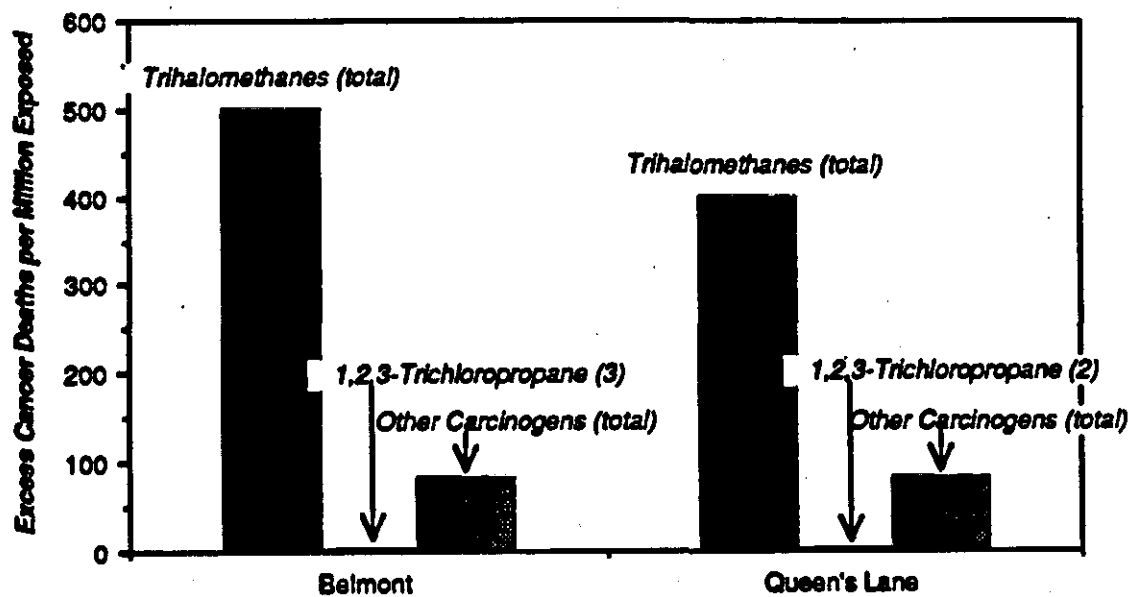
to be present from household consumption of water of this quality. TCP contributes <1% of the carcinogenic risk, while the other carcinogens as a group contribute 14%. For Queen Lane Treatment Plant effluent, the relative contributions to carcinogenic risk are as follows: trihalomethanes contribute 82%, TCP contributes <1%, and the other carcinogens contribute 17%.

6.5 Risk Perspective

An additional lifetime risk of cancer of 1×10^{-3} (i.e., 1 in a thousand) is estimated to result from household use of water containing the EPA's maximum allowable concentration (MCL) of chloroform. This estimate is obtained using the same assumptions as were utilized for estimating risks of TCP-contaminated water, and EPA's published potency factor for chloroform. It is important to understand that EPA's methodology for calculating cancer risk is based upon a set of conservative assumptions and does not provide a best estimate of risk but rather a probability that the risk will not exceed an upper limit. Other techniques for risk extrapolation have produced risk estimates of 4×10^{-5} or 1 in 25,000 for lifetime exposure to water containing EPA's limit of chloroform (Wilson and Crouch, 1987).

The lifetime risk of cancer from all causes is 0.20 to 0.25. That is, approximately 20 to 25 percent of all people develop cancer in their lifetimes. The estimated increase in lifetime risk of cancer due to daily exposure to TCP-contaminated water

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for 70 years is 5×10^{-6} using the 95% upper confidence limit
methodology based on 5 structurally related compoounds since the
NTP data are not presently available.* (See Appendix G). Thus,
for every 1 million persons exposed over a lifetime to unchanging
levels of TCP via household water use, 5 cases of cancer are
estimated to possibly result (Actually, there is a 95%
probability that fewer than 5 cases would develop due to TCP).
However, dividing by the 200,000 cancers from all other causes
expected in this population of 1 million leads to an
"insignificant" increase in cancer incidence of 0.0025% or less
(Wilson and Crouch, 1987).

6.6 Uncertainties in Cancer Risk

In examining substances for evidence of possible carcinogenic
effects on humans, EPA's Carcinogen Assessment Group (CAG) makes
two types of assessments: 1) a qualitative assessment of the
strength of the evidence suggesting that a chemical is
carcinogenic; and 2) where sufficient qualitative evidence of
carcinogenicity exists, a quantitative assessment of potency.

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* Different risk estimates using different extrapolation models
could provide a range of risk levels. However, the data which
would be needed to enable calculations using the Weibull,
Probit, Logit and other models have not been supplied to us.

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6.6.1 Uncertainties in Relying on Animal Cancer Tests for Human Prediction

Risk assessments rely on human epidemiological data where possible, but in most cases only data obtained in controlled animal studies is available. There is a large degree of uncertainty in extrapolating to diverse human beings data obtained from small, genetically homogeneous animals (often bred to be particularly susceptible to cancer or to spontaneously develop a very high incidence of cancer when no chemical carcinogen is administered.)

Of 392 chemicals in one data base, 96 were positive in the mouse and negative in the rat or vice versa (Ames, et. al., 1987). This raises some question about the reliability of the rodent model; if the mouse is not a very good predictor of carcinogenicity for the rat, how good is the rat as a predictor for man? Nevertheless, it has been used because it is practical and economically feasible.

6.6.2 Uncertainties in Extrapolating High-dose Animal Data to Very Low Exposures in Humans

Various models have been developed to perform these extrapolations. No single model is scientifically more valid than the others, nor are tests available to determine which is most accurate. Yet very large differences in risk can result from various models.

Superfund Public Health Evaluation guidelines rely on the linearized multistage extrapolation model (or a variant), which most scientists believe produces upper-bound estimates of low-dose risks. This model assumes that there is no threshold or

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dose without an effect; even a single molecule of a chemical can produce cancer, and the model predicts that risk is proportional to dose (linear relationship). Other extrapolation models are nonlinear, predicting that levels of risk drop more than proportionately as dose decreases. The non-linear models predict far lower risks at the doses encountered in the environment than does the one-hit model when applied to the same data. Risk estimates from the one-hit model are often hundreds or thousands of times higher than those derived from the alternative models. If animal cancer incidence data are available for more than one exposure level, EPA uses a "multistage" model. The estimation method assumes linearity at low doses even when the data indicate a non-linear relationship of known exposure levels.

The methods and assumptions used by EPA's Carcinogen Assessment Group (CAG) do not provide an expected value or best estimate of risk. Rather, the risk numbers generated provide an estimate of the plausible upper limit of risk. That is, the risk estimate is in reality a statistical expression which means that the true risk is unknown but there is a 95% probability that it is lower than the upper bound. In most cases the "plausible lower bound" is zero risk, as EPA's Carcinogen Assessment Group has pointed out. These wide differences between the linear models used by EPA and other models are precisely the differences that EPA's Science Advisory Board has pointed out as being needed to be laid out. It should also be noted that these models do not have a clear theoretical basis.

As EPA recognized in its cancer methodology paper, "Risk Evaluation Methods in the Santa Clara Valley Integrated Environmental Management Risk Assessment" (revised stage) report issued May 30, 1986:

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The procedures used by CAG, and followed by the IEMP, to calculate the possible carcinogenic impact of a substance on human beings are conservative in a number of ways. First, EPA uses a linear, non-threshold model to estimate the potency of a suspected carcinogen. This approach results in a fairly conservative (i.e., high) estimate of the potential impact of small doses of the substance. Second, in estimating the slope of the dose-response curve, CAG uses the upper-bound, 95% confidence estimate of the slope, rather than the best-guess or best-fit estimate. The use of this steeper dose-response curve is conservative in that it decreases the likelihood that we will underestimate the potency of a given substance. Other procedures ensuring that the CAG potency estimates are conservative are (1) the use of the most sensitive test species for extrapolating to humans; (2) the assumption that small doses will have an impact proportional to large (observed) test does; and (3) the use of a body surface area ratio (as opposed to a body weight ratio) in extrapolating from animal to human doses. For all these reasons, the potency scores developed by CAG and used by the IEMP should be regarded as plausible upper-bound estimates of potency.

EPA's Science Advisory Panel has urged use of maximum likelihood estimates (MLEs) in risk assessments performed in connection with pesticide regulations:

> Several of the EPA documents studied by the Panel in connection with the Registration Standards for pesticides include the following statement pertaining to the Agency's risk assessment procedures:

"The use of the upper 95% bound has been adopted
principally to provide assurance that the estimated
risks will not be materially affected by using the
results of any particular positive study."

The Panel disagrees with this approach. While it may
be mathematically satisfying, it is biologically
irrational and insensitive to reality. The upper 95%
confidence limit represents a linear extrapolation of
high dose data, when in fact non-linearities are
likely to be present at lower doses. Thus, it is not
scientifically valid to only present the upper 95%
bound.

The Panel reiterates its request that Agency risk
assessment discussions should include not only upper,
but also lower 95% confidence boundf values, and the
MLE (maximum likelihood estimate) in order to provide
information on the degrees of uncertainty involved in
any given risk assessment.

EPA's Science Advisory Board has similarly urged EPA to display
the results of more than one model as well as to display maximum
likelihood estimates and lower and upper confidence limits where
warranted by the data. As the SAB stated in comments on EPA's
draft Guidelines for Carcinogenic Risk Assessment:

It should be emphasized that the linearized
multi-stage model leads to a plausible upper limit
to the risk that is consistent with widely accepted
mechanisms of carcinogenesis. However, such an
estimate does not necessarily give a realistic
prediction of the risk. The true value of the risk

is uncertain, and for many substances the lower
bound estimate of risk is zero. The Agency's
procedures lead to a range of risk, defined by the
upper limit estimate from the linearized
multi-stage model and a lower limit; which should
be explicitly stated. An established procedure
that is applicable to a variety of substances does
not yet exist for making most likely or best
estimates of risk within the range of uncertainty
defined by the upper and lower limit estimates.
However, on a case-by-case basis where the data and
procedures are available, the Agency will strive to
provide most likely or best estimates of risk for
use in risk management. This will be most feasible
when human data are available and when exposures
are in the dose range of the data. . . . In
presenting quantitative estimates of risk and where
sufficient data are available, it will be
appropriate to provide the results of sensitivity
analyses of the various selected models used to
calculate risk. Such an analysis requires an
explicit statement of the assumptions and selected
parameters used in the models and will aid in
identifying the influence of changes in the
assumptions and parameters. This approach
clarifies those assumptions and parameters that are
most significant in influencing the final risk
estimate values.

Part of the problem with upper-bound assumptions is that even
modest overestimates can compound to yield a cumulative and
substantial exaggeration of the overall risk. The degree of

conservatism at each stage of the process is multiplied and accumulates to greatly exceed a best estimate of expected risk.

Some of the conservative assumptions utilized by CAG include:

- the use of the most sensitive test species for extrapolating to humans

- the assumption that small doses will have an impact proportional to large (observed) test doses

- the use of body surface area ratio (as opposed to body weight ratio) in extrapolating from animal to human doses

Because surface area increases by much less than weight as one moves from mouse to man, surface-area conversion leads to much higher risk estimates than weight conversion. Using mouse data the difference is roughly a factor of 13, and using rat data it is about a factor of 6.

In counting tumors in animal studies, both benign and malignant tumors are included, the argument being that benign tumors may progress to malignancy, and that a substance producing benign tumors in animals might produce malignant ones in humans. Most benign tumors, however, never become malignant.

The risk calculations also assume constant exposure on a daily basis for 70 years. Chemicals in the environment are subject to destruction by sunlight, microorganisms, and simple dilution and dispersion, to name a few of the fate processes that reduce contamination levels. Moreover, people move about from one area

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to another within the city each day, and are very likely to move from one city to another sometime during their lives.

Another major uncertainty is the relevance of data obtained from animals given massive doses of a chemical. The Office of Science and Technology Policy (OSTP) of the Executive Office of the President points out:

"These recommendations [for use of maximum tolerated doses] have been controversial because high doses may themselves produce altered physiological conditions which can qualitatively affect the induction of malignant tumors. Normal physiologic homeostasis and detoxification, or repair mechanisms may be overwhelmed and cancer which otherwise may not have occurred is induced or promoted."

Dosing animals to the point of severe toxic response may demonstrate that the "dose" is carcinogenic when the chemical is not. At the very low doses encountered by humans, the chemical may readily be detoxified and a threshold may exist for cancer-causing effects. Doses below the threshold would be without effect or risk. Thresholds may well exist for chemical carcinogenesis, but cannot be demonstrated experimentally. Thus, CAG has conservatively assumed that no threshold exists.

In conclusion, the propagation of worst-case conditions and assumptions in evaluating carcinogenic potential in humans and estimating potencies or risks at defined exposure levels leads to risk assessments that may greatly overestimate the true risk, if any. It is important to bear these considerations in mind when evaluating the extent of possible harm that could result from exposures to very small concentrations of chemicals.

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ACRONYMS

ACGIH	AMERICAN CONFERENCE OF GOVERNMENTAL AND INDUSTRIAL HYGIENISTS
ADI	ACCEPTABLE DAILY INTAKE
AIC	ACCEPTABLE INTAKE CHRONIC
AIS	ACCEPTABLE INTAKE SUBCHRONIC
BIBRA	BRITISH INDUSTRY BIOLOGICAL RESEARCH ASSOCIATES
CAG	CARCINOGEN ASSESSMENT GROUP - US EPA
CDI	CHRONIC DAILY INTAKE
CERCLA	COMPREHENSIVE ENVIRONMENTAL RESPONSE, COMPENSATION, AND LIABILITY ACT
CNS	CENTRAL NERVOUS SYSTEM
CT	CONCENTRATION TIMES THE TOXICITY CONSTANT FOR EACH MEDIUM
DBP	DIBUTYL PHTHALATE
DEHP	DI ETHYLHEXYL PHTHALATE
DNA	DESOXYRIBONUCLEIC ACID
EA	ENDANGERMENT ASSESSMENT
ECAO	ENVIRONMENTAL CRITERIA AND ASSESSMENT OFFICE - US EPA
ECEEC	EUROPEAN CHEMICAL INDUSTRY ECOLOGY AND ECOTOXICOLOGY CENTER
EPA	ENVIRONMENTAL PROTECTION AGENCY
FAO	FOOD AND AGRICULTURAL ORGANIZATION
Hc's	HENRY'S LAW CONSTANTS.
HSL	HAZARDOUS SUBSTANCE LIST
IARC	INTERNATIONAL AGENCY FOR RESEARCH ON CANCER
IRLG	INTERAGENCY REGULATORY LIASON GROUP
IS	INDICATOR SCORE
LD50	LETHAL DOSE WHERE 50% OF POPULATION DIES FROM AN IMPOSED-CAUSE.
LOAEL	LOW OBSERVED ADVERSE EFFECT LEVEL
MCL	MAXIMUM CONCENTRATION LEVEL
MED	MINIMUM EFFECTIVE DOSE
MPD	MINIMUM PREMARKETING DATA
MTD	MAXIMUM TOLERATED DOSE
NAAQS	NATIONAL AMBIENT AIR QUALITY STANDARDS
NAS	NATIONAL ACADEMY OF SCIENCE
NC	NONCARCINOGEN
NCAB	NATIONAL CANCER ADVISORY BOARD
NCI	NATIONAL CANCER INSTITIUTE
NCP	NATIONAL OIL AND HAZARDOUS SUBSTANCES POLLUTION CONTINGENCY PLAN
NIOSH	NATIONAL INSTITUTE OF SAFETY AND HEALTH
NOAEL	NO-OBSERVED ADVERSE EFFECT LEVEL
NOEL	NO-OBSERVED EFFECT LEVEL
NTP	NATIONAL TOXICOLOGY PROGRAM
OECD	ORGANIZATION FOR ECONOMIC COOPERATION AND DEVELOPMENT
OERR	OFFICE OF EMERGENCY AND REMEDIAL RESPONSE US EPA
OMB	OFFICE OF MANAGEMENT AND BUDGET
ORD	OFFICE OF RESEARCH AND DEVELOPMENT
OSHA	OCCUPATIONAL SAFETY AND HEALTH ADMINISTRATION
OSTP	OFFICE OF SCIENCE AND TECHNOLOGY POLICY
PAH	POLYCYCLIC AROMATIC HYDROCARBON
PC	POTENTIAL CARCINOGEN
PCE	TETRACHLOROETHYLENE
RA	RISK ASSESSMENT
SCE	SISTER CHROMATID EXCHANGE
SDI	SUBCHRONIC DAILY INTAKE
STEL	SHORT-TERM EMISSION LEVEL
TCE	TRICHLOROETHYLENE
TICs	TENATIVELY IDENTIFIED COMPOUNDS
TLV	THRESHOLD LIMIT VALUE
TWA	TIME WEIGHTED AVERAGE
WHO	WORLD HEALTH ORGANIZATION

GLOSSARY

ADENOMAS	A BENIGN TUMOR OF A GLANDULAR STRUCTURE OR OF GLANDULAR ORIGIN
ALKYLATION	THE ACT OR PROCESS OF ADDING ALKYL GROUPS (-CH3, ETC)
ATHEROMAS	A TUMOR WITH MATTER RESEMBLING GRULL
BIOACCUMULATION	TO ACCUMULATE IN A PLANT OR ANIMAL
BIOASSAYS	DETERMINATION OF THE RELATIVE STRENGTH OF A SUBSTANCE BY COMPARING ITS EFFECT ON A TEST ORGANISM WITH THAT OF A STANDARD PREPARATION
BLOOD DYSCRASIS	ABNORMAL CONDITION OF THE BLOOD
CARCINOGENICITY	THE ABILITY OF A SUBSTANCE TO PRODUCE CANCER
CARCINOGENS	A SUBSTANCE OR AGENT PORDUCING OR INCITING CANCER
CARCINOMAS	A MALIGNANT TUMOR OF EPITHELIAL ORIGIN
CHELATION	TO COMBINE WITH A METAL TO FORM A CHELATE RING
CHRONIC	LONG TERM, USUALLY 70 YEARS
CORPORA STRIATA	CORPUS (ONE OF THE CONDITIONS OF THE CORPUS LATIDA)
CYTOTOXICITY	DEGREE OF TOXICITY TO CELLS
DERMAL	SKIN
ECOTOXICOLOGICAL	TOXICOLOGY OF THE ENVIRONMENT
ENCEPHALOPATHIA	DISEASE OF THE BRAIN BY STRUCTURAL CHANGES
ENDOGENOUS	A CAUSE FROM INSIDE AN ORGANISM
EPIDEMIOLOGICAL	THE SUM OF FACTORS CONTROLLING THE PRESENCE OR ABSENCE OF DISEASE
EPIGENETIC	RELATED TO THE DEVELOPING OF NEW CHARACTERS IN AN UNDIFFERENTIATED ENTITY
ERYTHROCYTIC	DEALING WITH RED BLOOD CELLS
EXOGENOUS	A CAUSE FROM OUTSIDE AN ORGANISM
FETOTOXIC	TOXIC TO THE FETUS
GENOTOXIC	TOXIC TO THE GENES IN THE BODY
IN VITRO	OUTSIDE THE LIVING BODY AND IN AN ARTIFICIAL ENVIRONMENT
IN VIVO	IN THE LIVING BODY OF A PLANT OR ANIMAL
HEMANGIOENDOTHELIAL TUMORS	BENIGN TUMORS IN THE BLOOD VESSELS UNDER THE ENDOTHELIUM
HEPATOMA	MALIGNANT TUMOR OF THE LIVER
HEPATOTOXINS	TOXINS EFFECTING THE LIVER
HYPERKERATOSIS	EXCESSIVE DEVELOPMENT OF THE CORNEOUS LAYER OF THE SKIN
HYPOTHERMIA	SUBNORMAL TEMPERATURES OF THE BODY
HYDROLYSIS	CHEMICAL DECOMPOSITION INVOLVING SPLITTING A BOND AND THE ADDITION OF WATER
IMMUNOSUPPRESSIVE	SUPPRESSION OF NORMAL IMMUNE RESPONSES
INTRAPERITONEAL	WITHIN THE PERITONEUM
Koc	OCTANOL WATER PARTITION COEFFICIENT
LASSITUDE	A CONDITION OF LISTLESSNESS
LIPOPHILIC	HAVING AN AFFINITY FOR LIPIDS OR FATS
MALIGNANT	TENDENCY TO PRODUCE DEATH OR DETERIORATION
METABOLITE	A PRODUCT OF METABOLISM
MITOGENIC	A SUBSTANCE WHICH INDUCES MITOSIS
MORPHOLOGICAL	PERTAINING TO THE FORM OF A PLANT OR ANIMAL AND THEIR RESPECTIVE PARTS
MUTAGENICITY	AN AGENT THAT TENDS TO INCREASE THE FREQUENCY OR EXTENT OF MUTATION
MYOCARDIA	DEALING WITH THE HEART MUSCLE
NEOPLASMS	A NEW GROWTH OF TISSUE SERVING NO PHYSIOLOGICAL FUNCTION, I.E. TUMOR
OFF-SITE	OPERABLE UNITS OUTSIDE THE FENCED AREA
ONCOGENICITY	THE CAPACITY TO INDUCE OR FORM TUMORS
ON-SITE	AREA WITHIN THE FENCE
ORGANELLES	A SPECIALIZED CELLULAR PART THAT IS ANALOGOUS TO AN ORGAN
PEROXISOMES	A CYTOPLASMIC CELL ORGANELLE CONTAINING ENZYMES FOR THE PRODUCTION AND DECOMPOSITION OF HYDROGEN PEROXIDES
PHARMACOKINETICS	THE STUDY OF BODILY ABSORPTION, DISTRIBUTION, METABOLISM, AND EXCRETION OF DRUGS
PHOTOLYSIS	CHEMICAL DECOMPOSITIOIN BY THE ACTION OF RADIANT ENERGY
POLYNEUROPHATHY	MULTIPLE SYMPTOMS OF NEURO DEGENERACY
PORPHRIA	HEREDITARY ABNORMALITIES WHICH RESULT IN EXCESSIVE EXCRETION OF PORPHYRINS
REDOX	RELATED TO REDUCTION AND OXIDATION
SARCOMA	MALIGNANT NEOPLASMS OF THE CONNECTIVE TISSUE
SUBCHRONIC	SHORT TERM, USUALLY 10-90 DAYS
SYNERGISTIC	ADDITIVE
TERATOGENICITY	CONDITION OF FORMING MALFORMATIONS OR MONSTROSITIES
THIOL	THE GROUP SH CHARACTERISTIC OF MERCAPTANS
TRIGEMINAL NEUROPATHY	LOSS OF FUNCTION OF THE FIFTH CERVICAL NERVE

APPENDICES

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APPENDIX AA

EXPOSURE PATHWAYS FOR CONTAMINATED SOILS

Plate 1
Potential Exposure Pathways
From Contaminated Soils
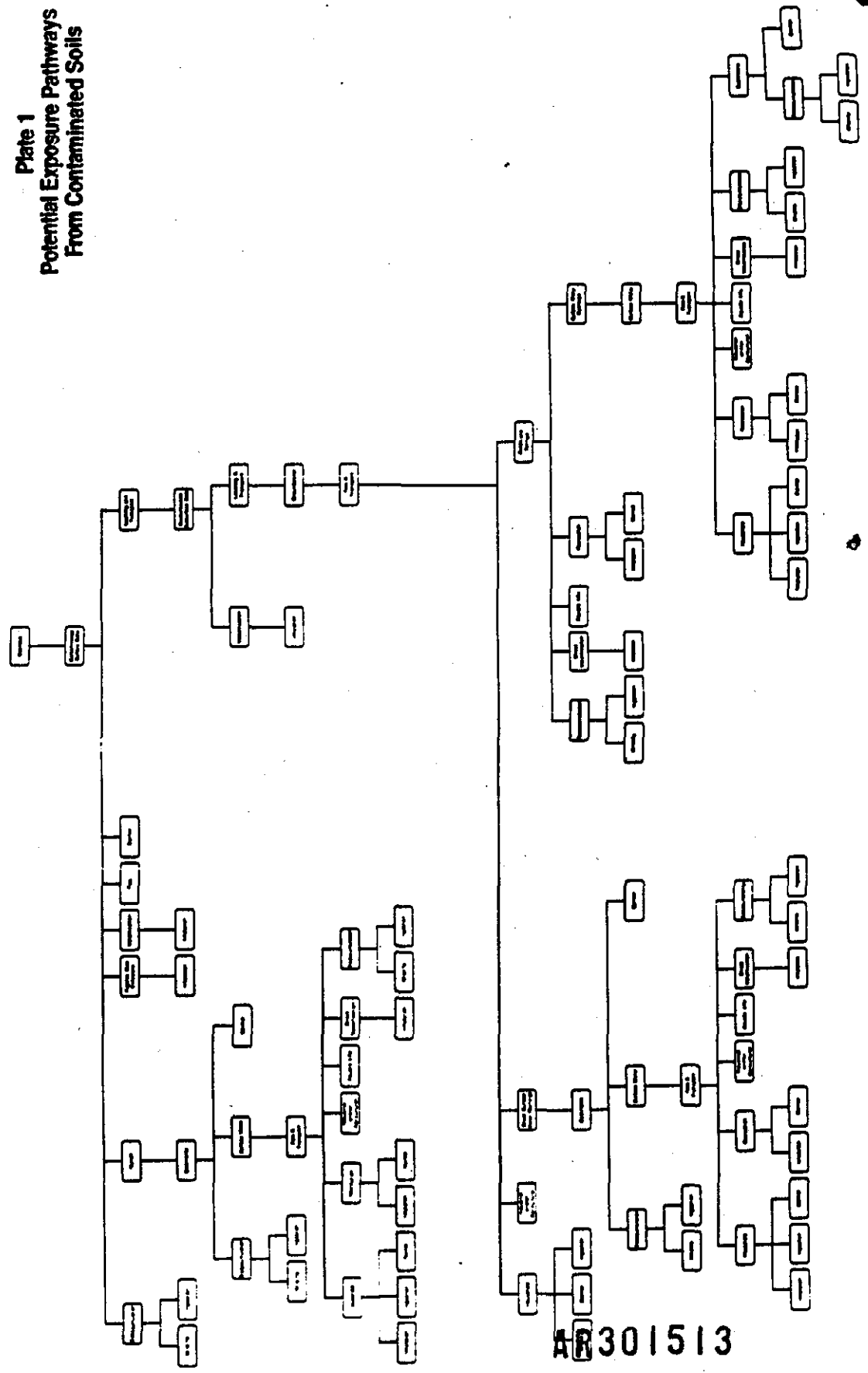
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APPENDIX A

EPA AND IARC
APPROACHES TO
CARCINOGENICITY STUDIES

APPENDIX A

The EPA has made the following modifications of the IARC approach
to classifying human and animal studies. For human studies:

1.   The observation of a statistically significant
     association between an agent and life threatening
     benign tumors in humans is included in the evaluations
     of risk to humans.

2.   A "no evidence" category is added. This category
     indicates that no association was found between
     exposure and increased risk of cancer in
     well-conducted, well-designed, independent analytical
     epidemologic studies.

For animal studies:

1.   An increased incident of combined benign and malignant
     tumors will be considered to provide sufficient
     evidence of carcinogenicity if the other criteria
     defining the "sufficient" category of evidence are met.

2.   An increased incident of benign tumors alone as
     "limited" evidence of carcinogenicity is added.

3.   Under specific circumstances, such as the production of
     neoplasms that occur with high spontaneous background
     incident, the evidence may be decreased to "limited" if
     warranted.

4.   A "no evidence" category is also added.

Agents that are judged to be in the EPA Weight-of-Evidence
stratification Groups A and B are to be regarded as suitable for
quantitative risk assessments. The appropriateness of
quantifying the risks from agents in Group C, specifically agents
that are at the boundary of Group C and D, would be judged on a
case-by-case basis. Agents that are judged to be in Groups D and
E should generally not be evaluated using quantitative risk
assessments.

Evidence of carcinogenicity from human studies comes from three
main sources:

1.   Case reports of individual cancer patients who were
     exposed to the agent(s).

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2. Descriptive epidemological studies

3. Analytical epidemologic (case control and cohort) studies.

Three criteria must be met before a causal association can be inferred between exposure and cancer in humans:

1. There is no identified bias which can explain the association.

2. The possibility of confounding has been considered and ruled out as explaining the association.

3. The association is unlikely to be due to chance. The degrees of evidence for carcinogenicity from studies in humans can be categorized by:

   a. Sufficient evidence of carcinogenicity, which indicates that there is a causal relationship between the agent and human cancer.

   b. Limited evidence of carcinogenicity, which indicates that a causal interpretation is credible.

   c. Inadequate evidence.

      i. There were few pertinent data, or

      ii. The available studies, while showing evidence of association, did not exclude chance, bias or confounding.

4. No evidence.

5. No data.

Assessment of evidence for carcinogenicity from studies in experimental animals are classified into five groups:

1. Sufficient evidence of carcinogenicity, which indicates an incident of malignant tumors or combined malignant and benign tumors:

   a. In multiple species or strains; or

   b. In multiple experiments (preferably with different routes of administration or using different dose levels); or

    c.   To an unusual degree with regard to incidence, site or type of tumor, or age at onset.

2.   Limited evidence of carcinogenicity.

    a.   Studies involve a single species, strain, or experiment; or

    b.   The experiments are restricted by inadequate dose levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or

    c.   An increase in the incident of benign tumors only.

3.   Inadequate evidence.

4.   No evidence.

5.   No data.

The categorization of overall evidence of carcinogenicity is subdivided into five groups.

    Group A: Human carcinogens are used only when there is sufficient evidence from epidemologic studies to support the causal association between exposure to agent(s) and cancer.

    Group B: Probable human carcinogens include agents for which the evidence of human carcinogenicity from epidemologic studies ranges from almost "sufficient" to "inadequate". B1 is reserved for agents for which there is at least limited evidence of carcinogenicity to humans from epidemologic studies. The agents for which there is inadequate evidence from human studies but sufficient evidence from animal studies would usually result in a classification of B2.

    Group C: Possible human carcinogens are used for agents with limited evidence of carcinogenicity in animals in the absence of human data. It includes a wide variety of evidence:

        a.   Definitive malignant tumor response in a single well-conducted study,

        b.   Marginal tumor responses in studies having inadequate design for reporting,

c.   Benign but not malignant tumors with an agent
     showing no response in a variety of short-
     term tests for mutagenicity, and

d.   Marginal responses in a tissue known to have
     a high and variable background rate.

Group D: Not classified is used for agent(s) with
          inadequate animal evidence of carcinogenicity.

Group E: No evidence of carcinogenicity for humans is used
          for agent(s) that shows no evidence for
          carcinogenicity in at least two adequate animal
          studies in different species or in both
          epidemologic animal studies.

The text for the general weight-of-evidence discussion is taken
from proposed guidelines for carcinogen risk assessment (1).

The Carcinogen Assessment Group (CAG) has evaluated fifty-four
chemicals as suspect human carcinogens and developed relative
carcinogenic potency factors for each chemical. The ranking of
potency indices is subjected to the uncertainty of comparing
different routes of exposure and a number of different species.
These indices are based on estimates of low dose risk using
linear multistage extrapolation from the observed range. Thus,
these indices are not valid when compared to potencies in the
experimental or observational range, especially, if linearity
does not exist in this range.

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APPENDIX B

WORKSHEETS USED TO SELECT
THE INDICATOR CHEMICALS FOR
TYSON'S SITE OFF-SITE OPERABLE UNITS RI
MONTGOMERY COUNTY, PA

WORKSHEET 1.
SCORING FOR INDICATOR CHEMICAL SELECTION:
CONCENTRATIONS AND Koc VALUES
IN VARIOUS ENVIRONMENTAL MEDIA
OPERABLE UNITS 1 - 5

NAME OF SITE: TYSON'S OFF-SITE
DATE PREPARED: 30 JULY 1987
PREPARED BY: T. A. SCHULLER

CHEMICAL	CAS NO	Koc VALUE	SEEP AREA SURFACE SOILS (mg/kg) RANGE	MEAN	GROUND WATER (shallow) (mg/L) RANGE	MEAN	SURFACE WATER (mg/L) RANGE	MEAN	FLOODPLAIN AREA SURFACE SOILS (mg/kg) RANGE	MEAN
Acetone	87-64-1	2.2								
Aluminum U	7440-36-0		BK		BDL - 944	69.276	BDL - 0.06	0.323	BK	
Antimony	7440-36-0		BK				BDL - 0.02	0.006		
Arsenic	7440-38-2		BK		BDL - 0.99	0.082	BDL - 0.011	0.003	BK	
Barium	7440-39-3		BK		BDL - 1.7	0.943	BDL - 0.4	0.115	BK	
Benzene	71-43-2	89			BDL - 0.057	0.00825				
Beryllium U	7440-41-7		BK		BDL - 0.012	0.001	BDL - 0.005	0.002	BK	
Bis(2-ethylhexyl)phthalate	117-81-7		BDL - 0.3	0.0138	BDL - 0.025	0.004	BDL - 0.026	0.003	BDL - 2.3	0.531
Cadmium	7740-43-9		BDL - 3.7	0.344	BDL - 0.039	0.012	BDL - 0.057	0.000304	BDL - 0.26	0.027
Chlorobenzene	108-90-7	330							BDL - 0.011	0.000786
Chloroform	67-66-3	31			BDL - 0.056	0.064	BDL - 0.01	0.004		
Chromium	7440-47-3		BK		BDL - 0.333	0.046	BDL - 0.05	0.018	BK	
Cobalt U			2.2 - 15.4	4.36	BDL - 0.344	0.067	BDL - 0.05	0.017	13 - 450	71.6
Copper	7440-50-8		BK				BDL - 0.1	0.00217		
Cresol	1319-77-3	500	BDL - 0.99	0.0288						
Di-n-butyl phthalate	84-74-2	170000								
1,3-Dichlorobenzene	95-50-1	1700							BDL - 0.9	0.08
1,2-Dichlorobenzene		1700			BDL - 0.033	0.0008			BDL - 0.9	0.127
1,4-Dichlorobenzene	106-46-7	1700			BDL - 0.008	0.00194	BDL - 0.01	0.000346	BDL - 1.8	0.193
1,1-Dichloroethane	75-34-3	30			BDL - 0.009	0.00282			BDL - 0.02	0.00133
1,2-Dichloroethane	107-06-2	14			BDL - 0.024	0.003				
Trans-1,2-dichloroethene	540-59-0	59			BDL - 0.095	0.0108			BDL - 0.04	0.00267
Methylene chloride	75-09-2	8.8	BDL - 0.019	0.00839			BDL - 0.005	0.0002	BDL - 0.11	0.0294
1,2-Dichloropropane	78-87-5	51								
1,3-Dichloropropene (cis & trans)	542-75-6	46			BDL - 0.00755	0.000844				
Diethyl phthalate	84-66-2	142								
Ethylbenzene	100-41-4	1100			BDL - 0.086	0.012	BDL - 0.002	4.17E-05	BDL - 0.57	0.0426
Iron U	15438-31-0		BK		BDL - 385	108.7	0.1 - 14.2	1.779	BK	
Lead	7439-92-1		BK		BDL - 0.795	0.186	BDL - 16.4	1.518	47 - 552	111
Manganese U	7439-96-5		BK		BDL - 16.4	4.9	BDL - 3.75	0.569	BK	
Mercury	7439-97-6		BK		BDL - 0.0003	0.0001	BDL - 0.0002	1.875E-05		
2-Butanone (Methyl ethyl ketone)	78-93-3	4.5								
4-Methyl-2-pentanone	108-10-1									
Nickel	7440-02-0		BK		BDL - 0.384	0.076	BDL - 0.04	0.013	BK	
N-Nitrosodiphenylamine									BDL - 0.56	0.0529
N-Nitrosodipropylamine										
Phenol	108-95-2	14.2					BDL - 0.008	0.000174		
Selenium	7782-49-2		BK		BDL - 0.003	0.00136	BDL - 0.002	0.001	BDL - 2.3	1.26
Silver	7440-22-4		BK				BDL - 0.0002	1.875E-05		
1,1,2,2-Tetrachloroethane	79-34-5	118								
Tetrachloroethene	127-18-4	364			BDL - 0.008	0.00224	BDL - 0.012	0.00023	BDL - 0.05	0.00567
Thallium U	7440-28-0						BDL - 0.01	0.00019		
Toluene	108-88-3	300			BDL - 0.19	0.0296	BDL - 0.084	0.0013	BDL - 0.22	0.0292
1,2,4-Trichlorobenzene	120-82-1	9200					BDL - 0.026	0.00046	BDL - 3.2	0.301
Trichloroethene	79-01-6	126			BDL - 0.007	0.00113	BDL - 0.016	0.0003	BDL - 0.04	0.00313
Fluorotrichloromethane U	75-69-4	2.53	BDL - 0.00755	0.000861					BDL - 0.011	0.00275
1,2,3-Trichloropropane U					BDL - 0.73	0.099	BDL - 1.7	0.139	BDL - 6.3	0.465
Vanadium	7440-62-2		BK		BDL - 0.427	0.16	BDL - 6.2	0.063	BK	
1,1,1-Trichloroethane									BDL - 0.077	0.0119
Total xylenes	1330-20-7	240			BDL - 0.14	0.0088	BDL - 0.19	0.0036	BDL - 1.8	0.18
Zinc	7440-66-6		BK		BDL - 0.095	0.392	BDL - 0.09	0.035	41 - 3070	331
Tin U					BDL - 0.479	0.07	BDL - 0.03	0.007	BDL - 21	4.49
2-Hexanone U										
2,4-Dimethylphenol U										
Di-octyl phthalate U							BDL - 0.042	0.00079		
Benzoic acid U									BDL - 25	3.14
Vinyl chloride										
Carbon disulfide							BDL - 0.0054	0.000113		

U - Unknown (no toxicity constant available) therefore, compound is not considered in the indicator chemical selection process
BK - Within background range for Eastern U.S. soils and therefore, is not considered in the indicator chemical selection process
BDL - Below detection limits

	SEDIMENTS (mg/kg)			SURFACE WATER (mg/L)			HILLSIDE AREA SEDIMENTS			SURFACE SOILS		
CHEMICAL	RANGE		MEAN	RANGE		MEAN	RANGE		MEAN	RANGE		MEAN
Acetone												
Aluminum U	1650	-	7640	BDL	-	0.554		4030	-	4030	4030	4650	-	119007	8020
Antimony												
Arsenic	9	-	15	7.37				6.3	-	6.3	6.3	BK		
Barium	31	-	432	139	0.130	-	0.130	0.130	55	-	55	55	BK		
Benzene	BDL	-	0.006	0.000214										
Beryllium U*	BDL	-	0.66	0.300				1.1	-	1.1	1.1	BK		
Bis(2-ethylhexyl)phthalate	BDL	-	19	0.782								BDL	-	0.47	0.0362
Cadmium	0.96	-	2.2	1.310	0.025	-	0.025	0.025	0.56	-	0.56	0.56	BDL	-	4.7	0.941
Chlorobenzene	BDL	-	0.66	0.012										
Chloroform	BDL	-	0.065	0.00223										
Chromium	3.1	-	46	17.6				4.4	-	4.4	4.4	BK		
Cobalt U	BDL	-	20	6.21				3.1	-	3.1	3.1	BK		
Copper	12	-	46	26.7				36	-	36	36	2.5	-	366	72.5
Cresol	BDL	-	14	0.946								BDL	-	1.4	0.145
Di-n-butyl phthalate	BDL	-	0.233	0.0117								BDL	-	0.29	0.029
1,2-Dichlorobenzene	BDL	-	1.09	0.0366										
1,2-Dichlorobenzene	BDL	-	4.5	0.25										
1,4-Dichlorobenzene	BDL	-	2.5	0.199										
1,1-Dichloroethane													6
1,2-Dichloroethane													
Trans-1,2-dichloroethene	BDL	-	0.071	0.00321										
Methylene chloride	BDL	-	0.045	0.081				0.013	-	0.013	0.013	BDL	-	0.0013	0.00545
1,2-Dichloropropane	BDL	-	0.055	0.001										
1,3-Dichloropropene (cis & trans)													
Diethyl phthalate													
Ethylbenzene	BDL	-	0.04	0.002										
Iron U	3630	-	19500	1099	0.138	-	0.138	0.138	7840	-	7840	7840	BK		
Lead	21	-	95	45.6								BK		
Manganese U	29	-	3240	664.7	0.035	-	0.035	0.035	905	-	905	905	BK		
Mercury	BDL	-	0.65	0.234										
2-Butanone (Methyl ethyl ketone)	BDL	-	0.11	0.00575										
4-Methyl-2-pentanone													
Nickel	4.6	-	29	11.4				3.7	-	3.7	3.7	BK		
N-Nitrosodiphenylamine													
N-Nitrosodipropylamine	BDL	-	2.5	0.0629										
Phenol	BDL	-	1.4	0.05										
Selenium												BK		
Silver													
1,1,2,2-Tetrachloroethane													
Tetrachloroethene	BDL	-	0.077	0.005	BDL	-	0.006	0.003					BDL	-	0.03	0.00321
Thallium U													
Toluene	BDL	-	0.98	0.0466										
1,2,4-Trichlorobenzene	BDL	-	44	1.76								BDL	-	0.021	0.00193
Trichloroethene	BDL	-	0.03	0.016	BDL	-	0.009	0.003					BDL	-	0.0075	0.00115
Fluorotrichloromethane U	BDL	-	0.537	0.029				0.0051	-	0.0051	0.0051	BDL	-	0.25	0.0361
1,2,3-Trichloropropane U	BDL	-	0.39	0.044	BDL	-	0.170	0.057	0.0092	-	0.0092	0.0092	BDL		
Vanadium	BDL	-	15	4.11								BK		
1,1,1-Trichloroethane													
Total xylenes	BDL	-	0.33	0.025										
Zinc	54	-	282	125	0.013	-	0.013	0.013	92	-	92	92	BK		
Tin U	BDL	-	14	6.99				7.9	-	7.9	7.9	7.9	-	29	3.87
2-Hexanone U	BDL	-	0.055	0.00204										
2,4-Dimethylphenol U												BDL	-	0.67	0.1
Di-octyl phthalate U													
Benzoic acid U	BDL	-	1.63	0.0582										
Vinyl chloride	BDL	-	0.016	0.000571										
Carbon disulfide													

U - Unknown (no toxicity constant available) therefore, compound is not considered in the indicator chemical selection process
BK - Within background range for Eastern U.S. soils and therefore, is not considered in the indicator chemical selection process
BDL-Below detection limits

CHEMICAL	SURFACE WATER			RAILROAD AREA SEDIMENT			SUBSURFACE SOILS			DEEP AQUIFER GROUND WATER		
	RANGE		MEAN	RANGE		MEAN	RANGE		MEAN	RANGE		MEAN
Acetone												
Aluminum U	0.226	-	0.252	0.238	1630	-	1840	1835	2760	-	14100	9044
Antimony												
Arsenic				3.1	-	5.4	4.25	BK				
Barium	0.106	-	0.14	0.123	34	-	64	49	BK			
Benzene												
Beryllium U*				BDL	-	0.46	0.23	BK				
Bis(2-ethylhexyl)phthalate												
Cadmium	0.002	-	0.028	0.015	0.47	-	0.67	0.67	BK			
Chlorobenzene								BDL	-	0.0071	0.0002	
Chloroform												
Chromium				2.1	-	9.7	5.6	BK				
Cobalt U				BDL	:	3.6	1.6	BK				
Copper				BDL	:	17	8.6	BK				
Cresol												
Di-n-butyl phthalate								BDL	:	0.22	0.007	
1,2-Dichlorobenzene								BDL	-	1.57	0.046	
1,3-Dichlorobenzene												
1,4-Dichlorobenzene								BDL	-	0.52	0.016	
1,1-Dichloroethane												
1,2-Dichloroethane												
Trans-1,2-dichloroethene												
Methylene chloride				BDL	-	0.025	0.020					
1,2-Dichloropropane												
1,3-Dichloropropene (cis & trans)												
Diethyl phthalate												
Ethylbenzene								BDL	-	0.025	0.001	
Iron U	0.076	-	0.807	0.293	3410	-	9040	5675	6330	-	28900	14116
Lead				9.9	:	75	42.25	BK				
Manganese U	BDL	-	0.059	0.030	94	:	165	139.5	BK			
Mercury												
2-Butanone (Methyl ethyl ketone)												
4-Methyl-2-pentanone												
Nickel				BDL	-	6	3	BK				
N-Nitrosodiphenylamine												
N-Nitrosodipropylamine												
Phenol												
Selenium												
Silver												
1,1,2,2-Tetrachloroethane												
Tetrachloroethene	BDL	-	0.012	0.006	BDL	-	0.029	0.014	BDL	-	0.14	0.0053
Thallium U												
Toluene								BDL	-	3.6	0.115	
1,2,4-Trichlorobenzene												
Trichloroethene	BDL	-	0.016	0.006	BDL	-	0.0094	0.0047	BDL	-	0.051	0.002
Fluorotrichloromethane U				BDL	-	0.005	0.002					
1,2,3-Trichloropropane U	BDL	-	0.93	0.265	BDL	-	0.036	0.019	BDL	-	0.15	0.010
Vanadium								BK				
1,1,1-Trichloroethane												
Total xylenes	BDL	-	0.006	0.004					BDL	-	0.13	0.010
Zinc	0.013		0.015	0.014	3.5	-	11	7.25	BK			
Tin U				BDL	-	3.5	1.75					
2-Hexanone U												
2,4-Dimethylphenol U												
Di-octyl phthalate U												
Benzoic acid U				BDL	-	16	8					
Vinyl chloride												
Carbon disulfide												

Deep Aquifer Ground Water column (partial readings):

CHEMICAL	RANGE			MEAN
Aluminum U	BDL	-	9.5	0.34
Antimony	BDL	-	0.01	0.0003
Arsenic	BDL	-	0.03	0.0008
Barium	BDL	-	5.41	0.61
Benzene	BDL	-	1.7	0.179
Chlorobenzene	BDL	-	2.4000	0.326
Chloroform	BDL	-	0.6	0.058
Chromium	BDL	-	0.6	0.0023
Cobalt U	BDL	-	0.02	0.0009
Copper	BDL	-	0.03	0.0009
Cresol	BDL	-	1.2	0.036
1,3-Dichlorobenzene	BDL	-	1	0.052
1,1-Dichloroethane	BDL	-	0.3	0.009
Trans-1,2-dichloroethene	BDL	-	0.13	0.004
1,2-Dichloropropane	BDL	-	0.1	0.003
1,3-Dichloropropene	BDL	-	34	3.12
Ethylbenzene	BDL	-	8.5	1.25
Iron U	BDL	-	22.16	0.81
Manganese U	BDL	-	12.4	0.43
Phenol	BDL	-	2.5	0.06
Tetrachloroethene	BDL	-	3.5	0.282
Thallium U	BDL	-	0.03	0.0006
Toluene	BDL	-	41	4.63
1,2,4-Trichlorobenzene	BDL	-	5	0.212
Trichloroethene	BDL	-	1.9	0.413
1,2,3-Trichloropropane U	BDL	-	1200	36.400
Total xylenes	BDL	-	64	8.240
Zinc	BDL	-	0.15	0.02
2-Hexanone U	BDL	-	22	1.86
2,4-Dimethylphenol U	BDL	-	0.3	0.009
Benzoic acid U	BDL	-	0.04	0.0015

U - Unknown (no toxicity constant available) therefore, compound is not considered in the indicator chemical selection process
BK - Within background range for Eastern U.S. soils and therefore, is not considered in the indicator chemical selection process
BDL-Below detection limits

WORKSHEET 1.
SCORING FOR INDICATOR CHEMICAL SELECTION:
CALCULATION OF CT AND IS VALUES FOR CARCINOGENIC EFFECTS
OPERABLE UNITS 1-3.

CHEMICAL	GROUNDWATER SHALLOW AND DEEP CT MAX	MEAN	SURFACE WATER CT MAX	MEAN	SURFACE SOIL CT MAX	MEAN	SUBSURFACE SOIL CT MAX	MEAN	SEDIMENTS CT MAX	MEAN	IS VALUE MAX	MEAN	TENTATIVE RANK MAX	MEAN
Arsenic and compounds	4.97E-02	2.82E-02	2.4E-02	1.22E-02					1.81E-03	1.21E-03	4.97E-02	2.82E-02	1	1
Benzene	9.06E-03	7.22E-04							2.38E-09	9.18E-11	9.06E-03	7.22E-04	4	5
Bis(2-ethylhexyl)phthalate (DEHP)									3.43E-07	2.27E-08	3.43E-07	2.27E-08	7	8
Cadmium and Compounds														
Chromium	2.3E-06	1.65E-08			3.0E-08	2.21E-08			1.0E-07	4.27E-08	2.3E-06	1.65E-08	2	3
Chromium VI and Compounds														
1,2-Dichloroethane (EDC)	7.0E-04	8.77E-05									7.0E-04	8.77E-05	5	6
Dichloromethane														
Nickel and Compounds					8.91E-08	8.35E-08			2.0E-04	9.18E-05	8.91E-08	9.35E-08	8	9
N-Nitrosodiphenylamine							6.1E-08	2.3E-08			2.0E-04	9.29E-05	6	7
N-Nitrosodi-n-propylamine							REF1	1.0E-08						
1,1,2,2-Tetrachloroethane														
Tetrachloroethene	1.9E-02	1.9E-03	9.4E-03	2.7E-05	1.7E-08	1.87E-08			2.3E-09	4.2E-09	1.9E-02	1.29E-03	3	4
Trichloroethene	4.0E-03	4.0E-03	5.8E-03	1.6E-05	6.5E-08	5.41E-10			4.0E-10	4.2E-08	2.2E-09	4.1E-03	9	2
1,2,3-Trichloropropane														

NAME OF SITE: TYSON'S OFF-SITE
DATE PREPARED: 30 JULY 1987
ANALYST: T. A. SCHULLER

CHEMICAL	GROUNDWATER CT		SURFACE WATER CT		SURFACE SOILS CT		SUBSURFACE SOILS CT		SEDIMENTS CT		IS VALUE		TENTATIVE RANK	
	MAX	MEAN	MAX	MEAN	MAX	MEAN	MAX	MEAN	MAX	MEAN	MAX	MEAN	MAX	MEAN
Acetone			8.11E-04	4.33E-04							8.11E-04	4.33E-04	25	23
Arsenic and Compounds														
Barium and Compounds														
Benzene														

*Detected in deep bedrock aquifer only. This operable unit is not included at this time.

AR301525

WORKSHEET 5
SCORING FOR INDICATOR CHEMICAL SELECTION
EVALUATION OF EXPOSURE FACTORS AND FINAL CHEMICAL SELECTION
OPERABLE UNITS 1 - 5

CHEMICAL	IS VALUE PC	RC	RANKING PC	NC	WATER SOLUBILITY (mg/L)	VAPOR PRESSURE (mm Hg)	HENRY'S LAW CONSTANT (atm-m3/mole)	Koc	HALF-LIFE (DAYS) GW	SW	SOIL	AIR	FREQUENCY DETECTED		IC
Acetone	9.08E-02	4.33E-04	1	23	1.00E+06	2.70E-02	2.06E-05	2.2		PERS		8.09	-	141	·
Arsenic and compounds		3.96E-08		11	NA	8.09E+00	NA	NA		PERS		4.09	70	113	·
Barium	7.22E-04	1.71E-09	6	2	NA	NA	NA	NA		1.00-1.00		8.09	108	113	·
Benzene	2.27E-06	1.10E-06	8	10	1.79E+03	8.95E-01	6.09E+00	83					6	141	·
Bis(2-ethylhexyl)phthalate (DEHP)		8.19E-08	·	8	NA	NA	NA	NA		PERS		4.09	84	119	
Cadmium and Compounds		2.02E-08		12	4.00E-02	1.17E-01	3.75E-09	330		0.3		3.90	17	141	
Chromium	1.09E-03		9		8.20E+03	1.51E-02	2.17E-08	31		0.3-330.0		80.00	4	113	· ·
Chromium					NA	NA	NA	NA		3.00-		4.09		113	
Chromium VI and Compounds		4.01E-08		10	3.10E-04	2.09E-01	1.19E-09	800		4.00-		8.09	95	141	
Copper and compounds		1.11E-01		8	1.30E-01	1.09E-05	2.09E-07	17000					37	141	·
Cobalt		3.01E-08		28	1.09E-03	1.09E-08	1.95E-09	1700		1.1-0.15		28.09	4	141	
Diethyl phthalate	7.91E-07			8									7	141	
1,3-Dichlorobenzene		2.11E-07		38	7.09E-01	1.19E+00	2.09E-08	1700		1.1-0.15		28.09	1	141	
1,4-Dichlorobenzene		8.09E-08		77	8.09E-03	1.09E-02	4.31E-08	38		1.3-0.8		46.09	1	141	
1,1-Dichloroethane		3.04E-08		28	6.30E-01	3.09E-02	6.09E-00	8.0		1.0-4.0		2.10	4	141	·
1,2-Dichloroethane (EDC)		7.04E-08		28	8.20E-00	6.09E-01	1.79E-04	14		0.17-		38-127	1	141	
1,2-Dichloroethene (cis & trans)		2.04E-08		31	2.09E-04	3.09E-02	2.03E-09	8.8		1.2-9.0		83.20	38	141	·
Dichloromethane		1.09E-04		24	2.70E-03	4.09E-01	2.31E-09	51		1.1-3.7		80.09	2	141	
1,3-Dichloropropene		8.19E-06	·	1									9	141	
Ethylbenzene		8.09E-00		18	1.99E-05	7.09E-00	6.09E-00	1100		1.6-7.6		1.46	28	141	
Lead and compounds (inorganic)	8.90E-08	9.60E-01	·	3	NA	NA	NA	NA		PERS		4.09	11	141	
Lead alkyl fumes**	8.37E-08	9.70E-08	·	25									84	113	·
Mercury and Compounds (inorganic)		1.09E-03		22	NA	NA	NA	NA		PERS		4.09	9	141	
Nickel and compounds		1.97E-01		8	NA	NA	NA	NA					28	141	
N-Nitrosodiphenylamine													84	119	·
Phenol	1.25E-03	4.01E-08	·	28	8.20E-04	3.41E-01	4.90E-07	142		8.02-9.0		0.05-9.0	3	141	·
Selenium and compounds		1.10E-01		7	NA	NA	NA	NA					90	113	·
Silver and compounds		1.11E-04	·	25	2.90E-03	8.09E-00	3.01E-08	118		0.4		894.00	43	113	·
Tetrachloroethene	1.25E-03	1.49E-08	4	21	1.50E-02	1.79E-01	2.90E-08	384		0.09		47.00	28	141	·
Toluene		1.21E-02		17	8.30E-02	2.01E+01	8.37E-09	300		0.17		1.30	18	141	·
1,2,4-Trichlorobenzene		2.27E-02		13	3.00E-01	2.90E-01	2.31E-09	900		1.2			12	141	·
Trichloroethene	4.09E-03	8.71E-01	2	4	1.11E-03	8.77E-01	9.10E-03	128		1.0-90.0		3.70	22	141	·
1,2,3-Trichloropropane													81	141	·
Vanadium and compounds		1.49E-08		18	NA	NA	NA	NA		1.5-4.0		8.00	85	113	·
Zinc and compounds		1.92E-02		16	1.95E-02	1.09E-01	7.04E-00	245		PERS		0.90	33	141	·
		2.26E-02		14	NA	NA	NA	NA				4.0-20	84	113	

These compounds were detected in method and travel blanks and were not included in the indicator chemical process. This unit is not being considered at this time due to further investigation and/or the RI.

APPENDIX C

ENVIRONMENTAL FATE AND TRANSPORT OF
THE INDICATOR CHEMICALS FOR
THE TYSON'S SITE OFF-SITE RI,
MONTGOMERY COUNTY, PA

APPENDIX C

C.1 Arsenic

Arsenic is a rare but ubiquitous element in the earth's crust with an average abundance of 5 ppm. Being the third member of Group VB of the periodic table, arsenic at times behaves much like phosphorus and antimony. In the natural environment, arsenic has four oxidation states of which the most common are +3 and +5. Because of the multiple oxidation states and arsenic's tendency to form soluble complexes, the geochemistry of arsenic is intricate and not well characterized, but the toxicology of many forms have been documented. Arsenic is generally encountered in copper smelting plants and is added to agricultural soils low in trace metals.

The major environmental fate processes of arsenic are sorption, bioaccumulation, and biodegradation/biotransformation. The cycling of arsenic through the environment is dominated by its sorption and desorption on soils and sediments. The half-life of arsenic in soils is given as persistent and is governed by the soil type, soil pH, phosphate levels, levels of iron or aluminum as well as residence in soil. Although some arsenic compounds are toxic, they do tend to bioaccumulated in lower levels of the food chain and to a certain extent in fish. Arsenic accumulates more readily in the fat rather than muscle tissue of fish with a half-life for arsenic in the gut and liver of green sunfish of seven days. Arsenic is readily biotransformed in aquatic environments to methylated forms. In these forms, arsenic becomes more mobile and enters the water and subsequently, the food chain. Thus, mobility could bring increasing concentrations of arsenic to the aquatic environment from contaminated sediments. Based upon the limited quantitative data available for arsenic, photolysis, oxidation, volatilization, and hydrolysis are considered to be environmentally insignificant fate process.

The environment transport pathway for arsenic is sorption onto soils, sediments, and/or suspended particles.

References: Callahan, M.A., et al, 1979; Mabey, W.R., et al., 1982; USEPA 1985b; Sittig, M., 1981, USEPA 1984d.

C.2 Barium

Barium occurs in nature chiefly as barite ($BaSO_4$) and in much smaller amounts as witherite ($BaCO_3$). The mineral forms are relatively insoluble in water, have high melting and boiling

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points, and very low vapor pressures. Barium is extremely reactive, decomposes in water, and readily forms insoluble carbonate and sulfate salts. Barium is generally present in solution in surface or ground water only in trace amounts. In aqueous solutions, barium always has a valence of +2 and combines readily with oxygen, nitrogen, hydrogen, ammonia, water, halogens, and sulfides. Some forms of barium are readily soluble in water whereas other forms are nearly insoluble. The solubility of barium compounds increases as the pH decreases. All of the barium salts are soluble in dilute acids except barium sulfate.

The predominant environmental fate process for barium is hydrolysis. Minor environmental significance is placed upon the importance of volatilization, biodegradation, hydrolysis, photolysis, oxidation, bioaccumulation, and sorption of barium due to the limited information available.

The major environmental transport process for barium appears to be volatilization due to fugitive dust emissions and/or dry fallout.

References: USEPA, May, 1986; Sittig, M., 1981; USEPA, September, 1985a; USEPA, September, 1985b.

C.3 Benzene

Benzene occurs naturally in the environment, but man-made inputs/releases have greatly increased its concentrations in the various media. Predominantly used as a starting material in the synthesis of organic chemicals, benzene is also used as a commercial solvent and in pesticides. A moderately volatile organic chemical with a high water solubility, benzene has a low chemical reactivity based upon the stability of the aromatic ring. From its density and water solubility, any benzene in excess of its water solubility would rise to the top of water, i.e., "floater".

The major environmental fate process is volatilization of benzene from both soil and water to the atmosphere. The volatilization half-life of benzene in water at 25°C has been calculated at 4.8 hours and an overall half-life in water estimated at 1-6 days. Once volatilized, benzene is available for oxidation by hydroxyl radicals (yielding phenol) and ozone. The atmospheric half-life of benzene in rural and urban settings is calculated to be 458 and 46 hours, respectively with an overall atmospheric half-life greater than 1 day. Sorption onto soils, sediments, and suspended particles occurs on a limited basis, thus, sorption is a less important fate process. Studies show that microorganisms in soil and water are capable of biodegrading benzene. However, this process is slow compared to the rate of volatilization.

Benzene is resistant to hydrolysis and photolysis. Little is known about the bioaccumulation of benzene, but based upon its octanol/water partition coefficient, it is anticipated to be very low.

The major environmental transport process for benzene is volatilization from soil and water to the atmosphere.

References: Haris, J. et al., 1981; Mabey, W.R. et al., 1982; Mills, W.B. et al., 1982; USEPA 1986; Gilbert, D. et al., 1980; Callahan, M.A. et al., 1979.

C.4   Toluene

Toluene is a flammable colorless liquid with a sour or burnt odor. It is moderately soluble in water but is miscible with most other organic solvents. Toluene occurs naturally as a component of petroleum oil and is produced indirectly in large volumes during gasoline refining and other operations. The main uses for toluene are as a raw material in the production of benzene and other organic solvents, as a solvent (especially for paints, coatings, gums, oils and resins), and as a gasoline additive to elevate octane ratings. This unsaturated aromatic hydrocarbon will float in water if its water solubility is exceeded, i.e., "floater".

The major environmental fate process for toluene is volatilization with an estimated half-life of 5.18 hours. Photooxidation is the primary atmospheric fate process for toluene with benzaldehyde as the principal organic product reported. Direct photolysis of toluene in the troposphere is energetically improbable while oxidation and hydrolysis in aquatic systems are probably not important. Little quantifiable information was found in the literature concerning the photolysis, hydrolysis, oxidation, biodegradation, and bioaccumulation of toluene in the environment. Therefore, these processes are considered to be of minor environmental significance. The biodegradation potential of toluene indicates that this compound would probably eventually degrade the environment, but not at a substantial rate.

The major environmental transport process for toluene is volatilization from soils or surface water (or both) to the atmosphere as well as fugitive dust emissions and dry deposition of toluene and oxidation products to the aquatic and terrestrial environments.

References: Callahan, M.A., et al, 1979; Mabey, W.R., et al, 1982; Mills, W.B., et al, 1982; Verschueren, K., 1983; USEPA, 1986; USEPA, September, 1985a and b.

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C.5 Trichloroethylene

Trichloroethylene (TCE) is ubiquitous in the environment although it is not naturally occurring. Widely used as a solvent in industrial degreasing of metals, TCE has minor uses in fumigant mixtures, inhalation anesthesia, and decaffeination of coffee. TCE is a highly volatile unsaturated aliphatic hydrocarbon with a relatively high water solubility. From its density, any TCE in excess of its water solubility would sink to the bottom of the water, i.e., "sinker".

Volatilization of TCE in the environment is the most important fate process with a laboratory half-life of 21 minutes. Once the compound enters the troposphere, high temperatures and UV radiation promote rapid degradation ($t_{1/2}$ = 4 days) to HCl, dichloroacetyl chloride, phosgene, carbon monoxide, and hexachlorobutadiene. The overall half-life of TCE in surface water and air is 1-90 days and 4 days, respectively. Since studies on the sorption properties of TCE onto sediments or suspended particles are limited, sorption will not be considered as a major fate process. TCE does not significantly bioaccumulate in the environment as seen by bioconcentration factors of 10-17 for bluegills with a half-life in tissue of less than 1 day. Biodegradation/biotransformation is of minor significance as an environmental fate process, however, higher mammals including man can degrade TCE to chlorinated acetic acids.

Environmental transport of TCE is due solely to volatilization from water to the atmosphere.

References: Callahan, M.A. et al, 1979; Mills, W.B. et al, 1982, USEPA 1985c.

C.6   1,2,4-Trichlorobenzene

1,2,4-Trichlorobenzene, a colorless crystalline substance with a characteristic aromatic acrid odor, has a low water solubility and relatively high vapor pressure. The major uses of 1,2,4-trichlorobenzene are solvents in chemical manufacturing; dyes and intermediate; dielectric fluid; synthetic transformer oils; lubricants; and insecticides. Based upon its density and water solubility, 1,2,4-trichlorobenzene will be classified as a "sinker" in water.

The major environmental fate processes for 1,2,4-trichlorobenzene are sorption, volatilization, and bioaccumulation. Although no quantitative sorption studies were found in the literature, the calculated value of the K_{OC} would indicate substantial sorption capabilities for 1,2,4-trichlorobenzene. Volatilization occurs rather rapidly from the water to the atmosphere with a calculated

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laboratory half-life of approximately 45 minutes. Once in the atmosphere, 1,2,4-trichlorobenzene is reported to be susceptible to attack by hydroxyl radicals (i.e. oxidation) with calculated half-life on the order of 1 to several days. 1,2,4-Trichlorobenzene has a high affinity for lipophilic materials and thus bioaccumulates in the aquatic environment. Insufficient evidence is available to determine whether 1,2,4-trichlorobenzene would biomagnify in the food chain. calculated half-life on the order of 1 to several days. Limited information exists concerning the photolysis and hydrolysis of 1,2,4-trichlorobenzene in the environment.

Environmental transport processes for 1,2,4-trichlorobenzene include sorption to soil/sediments and/or suspended particles and volatilization from soils or surface waters (or both) to the atmosphere.

References: Callahan, M.A., et al, 1979: Mabey, W.R., et al, 1982; Mills, W.B., et al, 1982, USEPA September 1985a and b, USEPA 1984a.

C.7   Chloroform

Chloroform is ubiquitous to the environment, both in urban and non-urban areas. Ninety percent of chloroform's use is in the production of chlorodifluoremethane with minor uses as a solvent, cleaning agent and fumigant ingredient. Chloroform, a dense, colorless, volatile liquid, is the most well known of the trihalomethanes. From its water solubility and density, any chloroform in excess of its water solubility would sink to the bottom of a water, i.e. "sinker".

The major environmental fate process is volatilization of chloroform from both soil and water to the atmosphere. The volatilization half-life of chloroform in water at 25°C has been calculated at 21 minutes and the overall half life in water estimated at 0.3 to 30 days. Due to the high vapor pressure of chloroform, volatilization into the atmosphere is quite rapid. Once in the troposphere chloroform is attached by hydroxyl radicals and form $CCl3$ radicals which react with oxygen to yield phosgene ($COCl_2$) and possibly chlorine oxide (ClO) radicals. These compounds further hydrolize to HCl and CO_2. Therefore, the primary fate process for chloroform once it has reached the troposphere is oxidation. Studies on adsorption, bioaccumulation, biotransformation/biodegradation of chloroform in the environment are limited in scope and therefore, these fate processes are considered of minor environmental significance. The log octanol/water partition coefficient of chloroform indicates a possible tendency of this compound to bioaccumulate under conditions of constant exposure. However, there is no evidence for biomagnification of chloroform in the aquatic food

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chain. The potential for biodegradation of chloroform in the
aquatic environment was examined and no aerobic biodegradation
was observed. However, studies conducted under anaerobic
conditions such as in lake sediments for ground water and studies
conducted in the presence of methanogenic bacteria, reported in
the reduction and degradation of chloroform under these
conditions. The quantity of information on biodegradation of
chloroform in the environment is limited and thus, biodegradation
is considered a minor environmental fate process.

References: Callahan, M.A., et al, 1979; Mills, W.B., et al,
1982; Mabey, W.R., et al, 1982; USEPA, 1985.

C.8 Xylene

Xylene is a flammable colorless liquid with a characteristic
sweet odor. It has limited solubility in water but is miscible
with most other organic solvents. Xylene occurs naturally as a
component of petroleum oil and is produced indirectly in large
volumes during gasoline refining and other operations. The main
uses for xylene are as a raw material in the production of
phthalic acid and anhydride and other specialty chemicals, as a
solvent (especially for paints, coatings, gums, oils and resins),
in the manufacture of dyes, insecticides, and pharmaceuticals,
and as a constituent in naphtha and asphalt. This unsaturated
aromatic hydrocarbon will float in water if its water solubility
is exceeded, i.e., "floater".

The major environmental fate process for xylene is votalization
with an estimated half-life of 5.18 hours. Biodegradation is a
primary fate process for xylene with toluic acid and methyl
catechol as the principal products reported. Direct photolysis
of xylene in the troposphere is energetically improbable while
oxidation and hydrolysis in aquatic systems are probably not
important. Little quantifiable information was found in the
literature concerning the photolysis, hydrolysis, oxidatioin, and
bioaccumulation of xylene in the environment. Therefore, these
processes are considered to be of minor environmental
significance.

The major environmental transport process for xylene is
votalization from soils or surface water (or both) to the
atmosphere as well as fugitive dust emissions and dry deposition
of xylene.

References: Callahan, M.A., et al, 1979; Mabey, W.R., et al,
1982; Mills, W.B., et al, 1982; Verschueren, K.,
1983; USEPA, 1986; USEPA, September, 1985a and b.

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C.9   1,2,3-Trichloropropane

1,2,3-Trichloropropane (1,2,3-TCP) is a colorless liquid with a strong acid odor. It is moderately soluble in water and is miscible with most other organic solvents. The main uses for 1,2,3-TCP are as a paint and varnish remover, a solvent, and a degreasing agent. This saturated aliphatic hydrocarbon will sink in water if its water solubility is exceeded, i.e., "sinker".

The fate and transport processes for 1,2,3-TCP are based upon a similar compound (1,2-dichloropropane) since very limited data is available for 1,2,3-TCP. The major environmental fate process for 1,2,3-TCP appears to be volatilization from surface soils and water. From the limited quantifiable data available, sorption appears to be moderate, bioaccumulation slight, and biodegradation very slow. Further information on photolysis, oxidation, and hydrolysis is not available and at this time these processes are considered to be of minor environmental significance.

The major environmental transport process for 1,2,3-TCP appears to be volatilization from soils or surface water (or both) to the atmosphere. Other transport processes would include fugitive dust and dry deposition of 1,2,3-TCP to the aquatic and terrestrial environments.

References: Callahan, M.A., et al, 1979; Mabey, W.R., et al, 1982; Mills, W.B., et al, 1982; Verschueren, K., 1983; USEPA, 1986; USEPA, September, 1985a and b.

C.10   Tetrachlorethene

Tetrachloroethene is a colorless liquid of importance in the organic chemical industry. It has many uses, including uses as a solvent, in dry cleaning operations, in metal degreasing, removing caffeine from coffee, and various manufacturing processes. Tetrachloroethene is a fairly ubiquitous chemical and has been detected in both fresh and marine systems, as well as in food and human tissues. When tetrachloroethene exceeds its solubility in water, it will sink, i.e., "sinker".

The major environmental transport process for tetrachloroethene is volatilization. The half-life for tetrachloroethene in surface waters has been reported to be approximately 20 to 27 minutes. Once in the atmosphere, tetrachloroethene is susceptible to attack at the double bond by hydroxyl radicals. The tropospheric lifetime has been estimated at 10 days. Photolysis is probably not an important fate process, as oxidation occurs so rapidly. Sorption is not an important fate process.   There is some evidence of bioaccumulation of

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tetrachloroethene by marine organisms, however there is no evidence of biomagnification up the food-chain. There is some evidence that biodegradation does occur, however it does not seem to occur at appreciable rates.

Overall, the most important environmental transport and fate processes for tetrachloroethene is volatilization out of soils and surface waters and hydroxyl radical attack in the troposphere.

References: Callahan, M.A., et al, 1979; Verschueren, K., 1983; Mackey, D. and P.S. Leinonen, 1975.

C.11   Ethylbenzene

Ethylbenzene is a colorless liquid that is produced in petroleum refining and the organic chemical industries. It has many uses as a solvent, in styrene and acetophenone manufacturing, and as an asphalt constituent. This aromatic hydrocarbon will float on water if its water solubility is exceeded, i.e., "floater".

The major environmental fate process for ethylbenzene is volatilization with a half-life approximated to be 5 to 6 hours. Data is lacking about most fate processes for ethylbenzene. Photooxidation would be expected to be the primary atmospheric fate process. Direct photolysis in the atmosphere is slow. A half-conversion time of approximately 15 hours has been reported for photolysis of ethylbenzene. Little or no data was found in the literature regarding oxidation, hydrolysis, bioaccumulation, or biodegradation of ethylbenzene in the environment. These processes are considered to be of minor environmental significance. Sorption would possibly be an important fate process for ethylbenzene based on its octanol/water partition coefficient. However, no specific environmental sorption data was available.

The major environmental transport process for ethylbenzene is volatilization from surface soils and/or surface water to the atmosphere. Overall, although since little data is available for ethylbenzene, based on its structure, it would be expected to behave similarly to toluene.

References: Callahan, M.A., et al, 1979; Verschueren, K., 1983; Mackey, D. and P.J. Leinonen, 1975.

C.12   Chlorobenzene

Chlorobenzene is a flammable, colorless liquid with an aromatic, mothball odor. It is almost immiscible in water (moderate-to-low water solubility) but is miscible with most other organic

solvents. Chlorobenzene's main use is in solvent recovery plants with minor uses in the manufacture of aniline, insecticides, phenol, and chloronitrobenzene. This unsaturated aromatic hydrocarbon will sink in water if its water solubility is exceeded, i.e. "sinker".

The major competing fate processes of chlorobenzene in the environment are sorption, volatilization, and bioaccumulation. Although no specific environmental sorption studies were found in the literature, the values for the log octanol/water partition coefficient and the sediment/soil partition coefficient indicate that sorption processes may be substantial for chlorobenzene. The overall half-life of chlorobenzene in the air and surface water is 3.5 days and 0.3 days respectively, while the volatilization half-life is on the order of 0.5 to 9 hours. Experimental evidence indicates that chlorobenzene has an intermediate potential for bioaccumulation in the lymphatic tissues of living organisms. However, no information is currently available upon chlorobenzene's biomagnification in the food chain. Little quantifiable information was found in the literature concerning the photolysis, hydrolysis and oxidation of chlorobenzene in the environment. Therefore, these processes are considered to be of minor environmental significance. The biodegradation potential of chlorobenzene indicates that this compound will probably eventually degrade in the environment, but not at a substantial rate.

The major environmental transport processes for chlorobenzene are volatilization from soils or surface water (or both) to the atmosphere and sorption to soils/sediments and/or suspended particles.

References: Callahan, M.A., et al, 1979; Mabey, W.R., et al, 1982; Mills, W.B., et al, 1982, Vershueren, K., 1983; USEPA 1985b, USEPA 1984a.

APPENDIX D

EXPOSURE PATHWAYS

Plate 2
Deep Aquifer Exposure Scenarios

ERM

Plate 3
Hillside Exposure Scenarios

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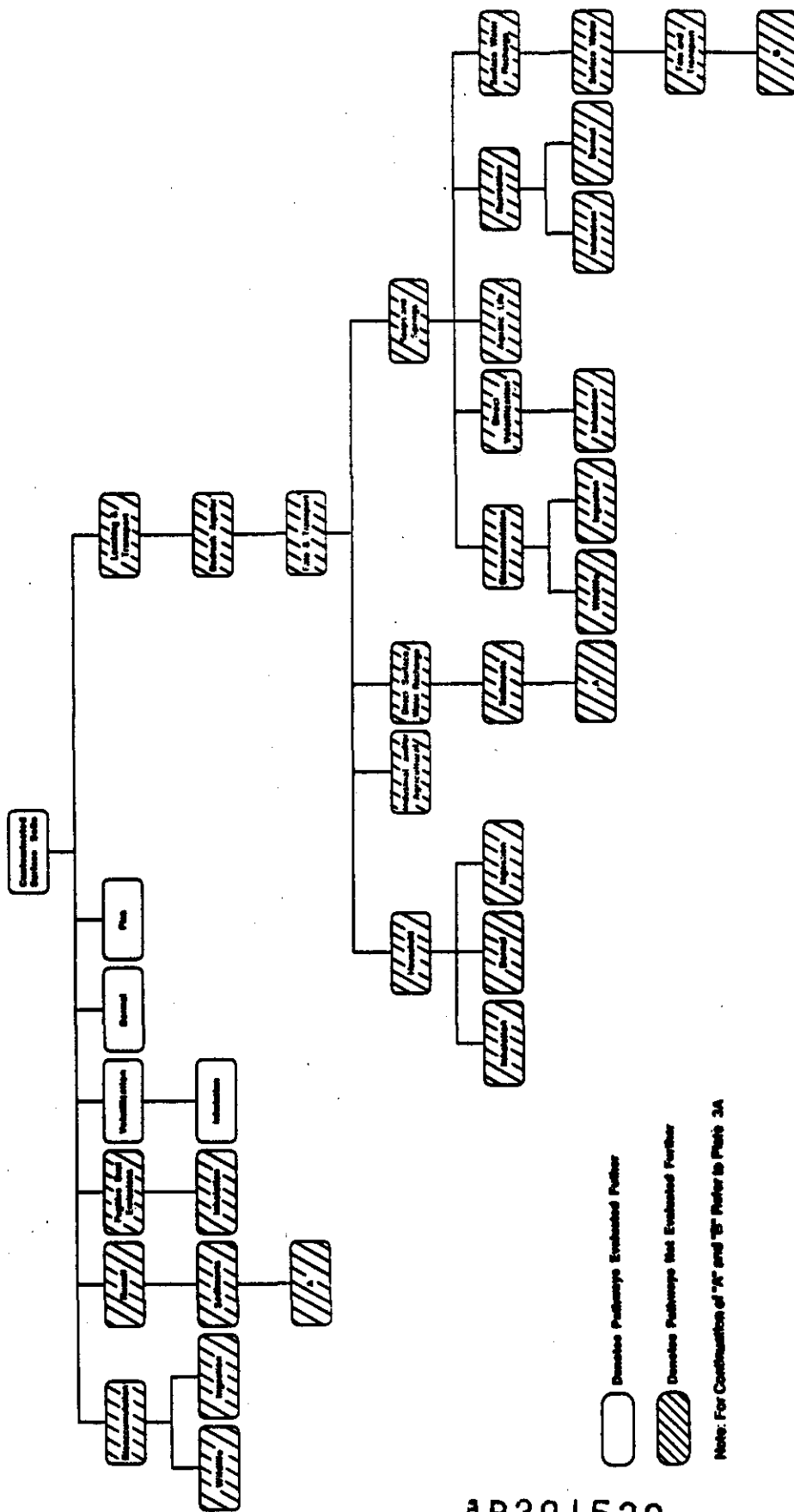
Plate 3A
Hillside Exposure Scenarios
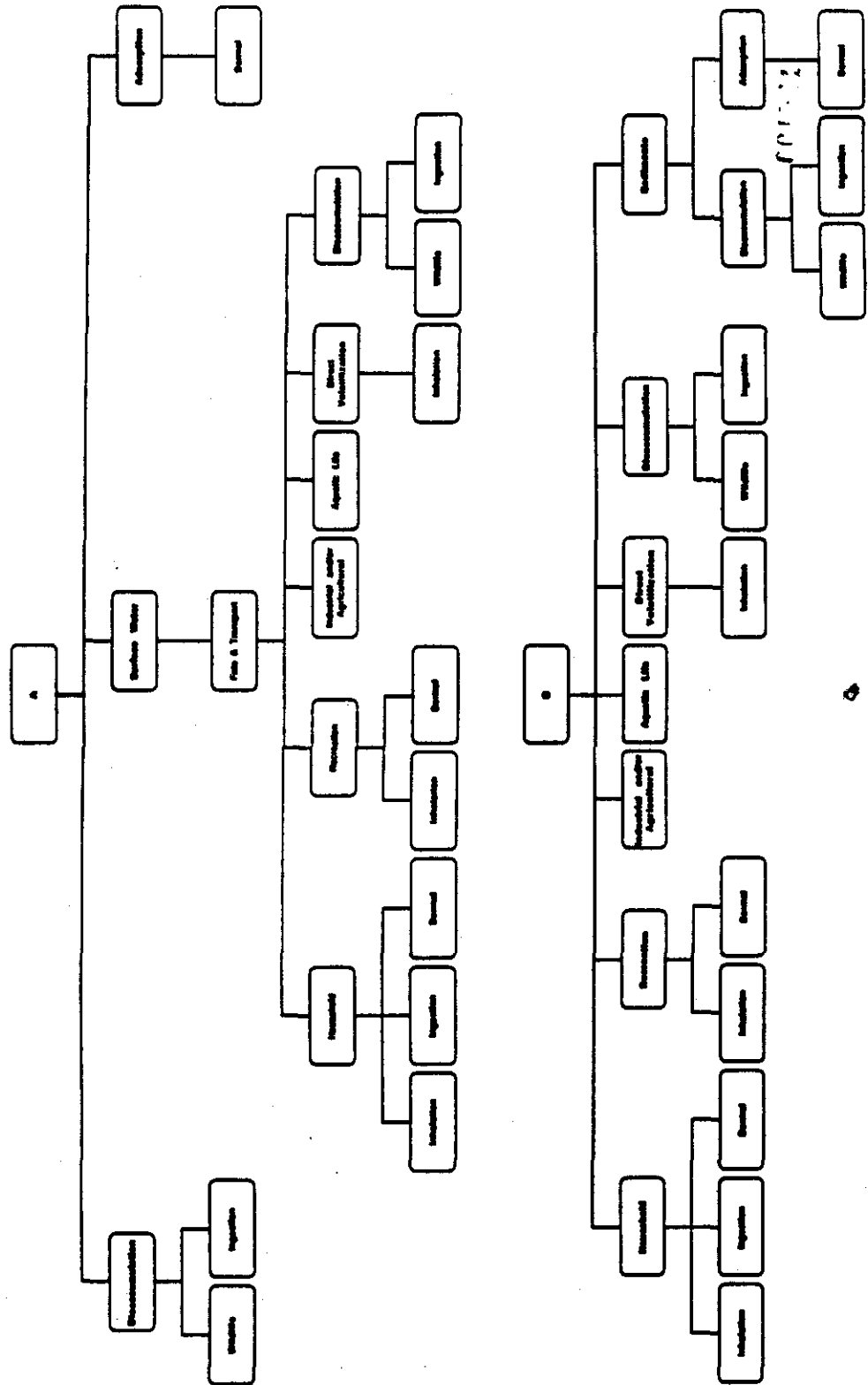
Plate 4
Railroad Exposure Scenarios
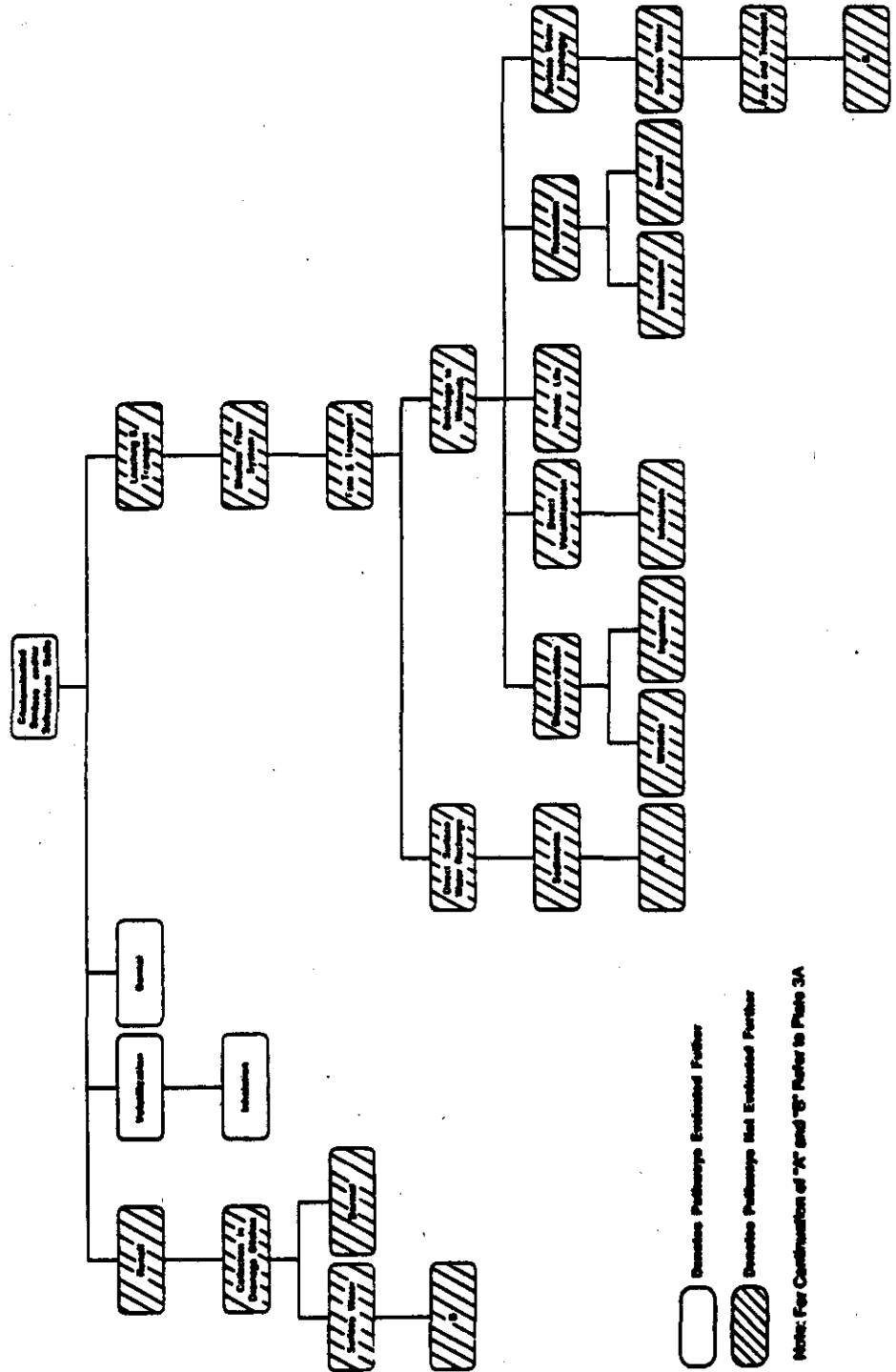
ERM

Plate 5
Seeps Exposure Scenarios

ERM
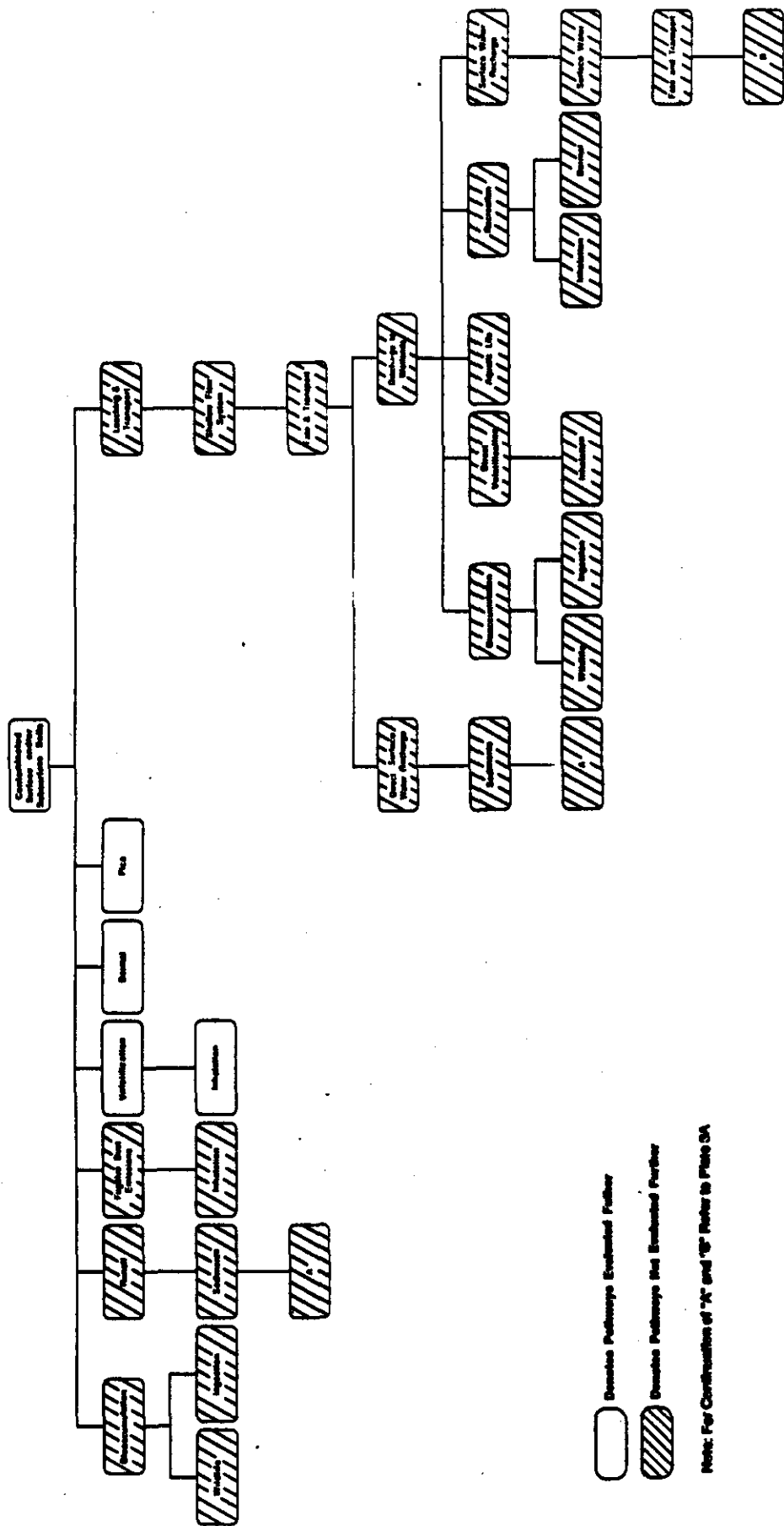
Plate 6
Flood Plain/Wetland
Exposure Scenarios
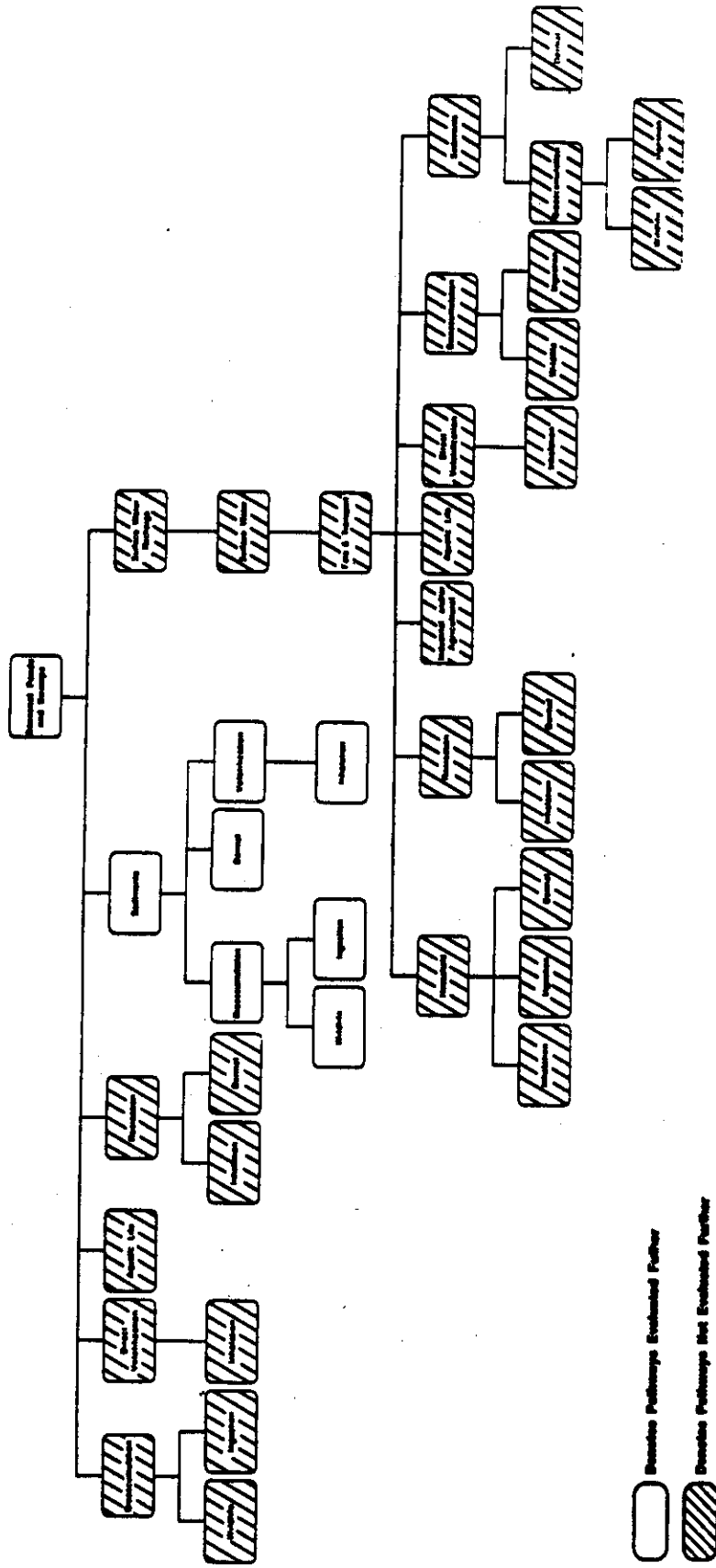
APPENDIX E

DATA USED IN THE
ENDANGERMENT ASSESSMENT

TABLE E-1
TYSON'S SITE
FLOODPLAIN AREA SHALLOW GROUND WATER RESULTS
INORGANIC CONSTITUENTS
mg/L

Well Number	NUS-7	001	002	001	001	002	002	NUS-7
Sample ID	GW035	GW037	GW036	940006	940160	940010	940161	940163
Date Sampled	A	A	A	1/4/84	2/28/84	1/4/84	2/28/84	2/28/84
Parameter								
Aluminum	0.03NV	0.06NV	0.07NV	39.6	29	244	82.8	160
Chromium			0.002NV	0.046	0.04	0.256	0.1	0.23
Barium	0.2	0.06J	0.04J	0.426	0.3	1.22	0.5	1.7
Beryllium				<0.005	<0.005	0.012	<0.005	0.01
Cobalt	0.005NV	0.005NV	0.02NV	<0.050	<0.050	0.233	0.05	0.1
Copper	0.0005NV	0.002NV	0.003NV	<0.050	<0.050	0.344	0.1	0.25
Iron	24.2	0.04B	0.05B	42.1	31.3	365	120	271
Nickel				0.04	0.04	0.264	0.06	0.2
Manganese	6.34	0.56	1.64	0.893	0.81	10	2.52	16.4
Arsenic	0.0096	0.0005NV	0.001NV	0.033	0.019	0.29	0.064	0.091
Antimony	0.001NV	0.002NV	0.001NV	<0.020	<0.020	<0.020	<0.020	<0.020
Selenium				<0.002	<0.002	0.003	<0.002	<0.002
Thallium	0.002NV	0.003NV	0.001NV	<0.010	<0.010	<0.010	<0.010	<0.010
Zinc	0.01B	0.01B	0.02B	0.144	0.11	0.695	0.28	0.45
Vanadium	0.02J		0.03J	<0.200	<0.200	0.427	<0.200	0.2
Silver				<0.010	<0.010	<0.010	<0.010	<0.010
Mercury				<0.0002	0.0003	0.00032	0.0003	0.0002
Tin	0.04B	0.028	0.025	0.061	NOS	0.478	NOS	NOS
Cadmium		0.0002NV	0.001NV	<0.001	<0.001	0.025	<0.001	0.003
Lead		0.001NV		0.124	0.025	0.795	0.062	0.26
TOX				0.052	0.148	0.124	0.145	0.63
TOX (Dup)				0.054		0.115		
pH (SU)				6.8	5.9	6.4	5.5	5.9
Temperature (°C)				6	6	10	6	6
Specific Conductance (umhos)				365	160	910	220	475
Data Prepared By:	EPM, Inc.	EPM, Inc.	EPM, Inc.	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA

A - From report dated 8 December 1986

NV - Not valid - This result is below the instrument detection limit demonstrated in the data.

Blank = none detected

B - This result is questionable since this compound/constituent was detected in blank(s) at similar levels.

J - estimated value

All EPM data has gone through a quality assurance review.

Well Number	NUS-7	001	002	001	001	002	002	NUS-7
Sample ID	GW035	GW037	GW036	840009	840160	840010	840161	840163
Date Sampled	A	A	A	1/4/84	2/28/84	1/4/84	2/28/84	2/28/84
Parameter								
VOLATILES								
1,2,3-Trichloropropane	0.73	0.022	0.013			0.029*		
1,1-Dichloroethane	0.006					LT		0.006
trans-1,2-Dichloroethene	0.005							
Trichloroethene	0.007							0.002K
Benzene	0.008		0.009			0.037		0.012
Tetrachloroethene	0.009			LT	0.0014K			
Toluene	0.013		0.006			0.18		0.02
Chlorobenzene	0.017		0.011			0.039		0.029
Ethylbenzene	0.031					0.066		
Total xylenes	0.061		0.009			0.14	LT	0.076
trans-1,3-Dichloropropene								LT
1,2-Dichloroethane								0.024
SEMI-VOLATILES								
1,4-Dichlorobenzene			0.005J			LT		
Bis (2-ethylhexyl) phthalate	0.009B							
PESTICIDES								
Endosulfan II			0.00002NC					
Data Prepared By:	ERM, Inc.	ERM, Inc.	ERM, Inc.	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA

A - From report dated 8 December 1986
Blank - none detected
* - or isomer
LT: Less than the specified detection limit but greater than one half of the detection limit (present but below detection limit)
K: Actual value is less than given value
NC - Not confident - The methods of analysis is susceptible to false positives.
All ERM data has gone through a quality assurance review.

TABLE E-2
TYSON'S SITE
FLOODPLAIN AREA SEDIMENT RESULTS
HSL INORGANIC CONSTITUENTS
mg/kg, dry weight

Sample Number	840050	840052	840054 Duplicate	840056	840060	840062	840064	840066	840068	840070** Background	840058** Background well
Date Sampled	1/9/84	1/9/84	1/9/84	1/9/84	1/10/84	1/10/84	1/10/84	1/10/84	1/10/84	1/10/84	1/10/84
Parameter											
Aluminum	3400	4700	3490	7550	7040	2130	3770	1080	3490	6110	1020
Chromium	5.4	9	6.6	30	21	42	6.2	6.1	23	24	2.1
Barium	31	62	73	67	106	86	112	73	62	86	17
Beryllium	0.36	0.72	0.63	0.68	0.68	0.68	0.38	<0.25	0.75	0.63	<0.25
Cobalt	5.1	6.8	6.1	20	17	7.8	5.1	<2.5	17	20	<2.5
Copper	15	12	13	48	32	18	23	30	38	43	1530
Iron	6110	13500	6140	10700	14400	14400	5430	9770	9900	10900	1530
Nickel	4.8	7.4	9	23	21	7	8.4	5.3	22	24	2.6
Manganese	79	546	400	658	3340	26	231	118	504	658	70
Magnesium											
Arsenic	9	6.2	3.1	6.6	14	7	6.4	6.1	15	10	3.8
Antimony	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1	<1
Selenium	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Thallium	34	108	91	148	154	292	108	83	130	144	24
Zinc	<10	<10	<10	12	15	<10	<10	<10	10	10	<10
Vanadium	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Silver	<0.1	0.7	0.05	0.1	0.1	<0.1	0.1	0.15	0.1	<0.1	<0.1
Mercury	7.1	5.1	5.4	14	13	<1	6.7	1.5	10	11	5.3
Tin	0.56	0.98	0.6	2.1	1.8	1.5	1.2	0.75	2.2	1.9	<0.05
Cadmium	21	32	32	69	95	23	54	54	63	78	11
Lead											
Calcium											
TOX	<100	<100	<100	<100	<100	<100	<100	<100	<100	<100	<100
Oil & Grease	<100	190	290	1500	<100	<100	<100	340	130	150	<100
Data reported by	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA

All EPA data have gone through a quality assurance review.
** Data not included in the calculation of average or range

TABLE E-2 (Continued)
TYSON'S SITE
FLOODPLAIN AREA SEDIMENT RESULTS
HSL INORGANIC CONSTITUENTS
mg/kg, dry weight

Sample Number	840161** Background unit	840072** Background unit	BA-001S	BA-002S	BA-003S	BA-004S	BA-005S	BA-006S	BA-007S	Weir 04 1475
Date Sampled	2/29/84	1/19/84	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	6/19/87
Parameter										
Aluminum	2600	2790	8490	17100	10400	8410	11900	8120	14400	7130
Chromium	2.5	2.6	9.4	27.3	27.4	13.4	14.7	10.2	21.2	31
Barium	45	44	72.2	452	227	104	100	64.8	136	225
Beryllium	<0.25	0.35	0.49 B	2.18	1.1	0.8	0.7	0.49 B	1.08	1.3
Cobalt	<2.5	<2.5	4	16.4	18.2	8	0.5	2.5	10.8	18
Copper	6	3.1	26.1	98.4	46.3	26.3	24.5	12.7	109	71
Iron	3930	3970	6960	39100.00	20900	18900	15200	7850	20000	18400
Nickel	2	2.6	5.3	21.9	21.9	10.4	8.2	8.9	13.3	24
Manganese	93.5	149	229	406	1120	142	492	135	347	1080
Magnesium										1850
Arsenic	1.7	4.3	2.54	13.7	8.2	4.3	5.22	2.8	11.4	10
Antimony	<1	<1								
Selenium	0.2	0.2	0.49 B	3	1.9	0.39 B	0.20 B	0.38 B	1.0 B	
Thallium	<0.5	<0.5		3.36		0.60 B	1.9 B	0.90 B	1.9 B	
Zinc	52	14	77.0	1070	290	96.2	96.1	91.7	152	251
Vanadium	<10	<10	12	90	43.6	11.9	19.6	11.4	37.1	18
Silver	<0.5	<0.5								
Mercury	EW	<0.1								
Tin	ND	5.4								
Cadmium	0.1	0.26		0.11	0.8	0.2	0.2		0.8	0.29
Lead	17	18	22.7	108	126.0	95.4	95.5	29.2	103	95
Calcium										2520
TOX	<100	<100	NA	NA	NA	NA	NA	NA	NA	NA
Oil & Grease			NA	NA	NA	NA	NA	NA	NA	NA
Data reported by:	Betor/TSA	Betor/TSA								

All ETM data have gone through a quality assurance review.

** Data not included in the calculation of average or range

TABLE E-3
TYSON'S SITE
FLOODPLAIN AREA SURFACE WATER RESULTS
HSL CONSTITUENTS
mg/L

SAMPLE ID	Site G Non-filtered	Site G Filtered	840043	840045	840049	840051	840053 duplicate	840055	840059	840061	840063	840065	840067	840069 '' Background	840047
Sample Location															
Date Sampled	A	A	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84	1/9/84
Parameter															
Aluminum	0.5		0.226	0.252	<0.2	<0.2	<0.2	0.957	0.294	0.249	0.241	0.270	0.257	0.243	<0.200
Chromium	0.018	0.048	<0.010	<0.010	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.010
Barium			0.14	0.105	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	<0.1	0.12
Beryllium			<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005
Cobalt	0.03		<0.050	<0.050	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.050
Copper	0.05	0.1	<0.050	<0.050	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.05	<0.050
Iron			0.078	0.507	0.1	0.281	0.270	1.51	0.118	1.69	0.2	14.2	0.378	0.28	0.128
Nickel			<0.040	<0.040	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04	<0.04	<0.040
Manganese	0.13	0.11	0.010	0.059	0.021	0.042	0.042	0.148	<0.01	0.104	0.018	2.28	0.252	0.229	0.026
Arsenic			<0.010	<0.010	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.010
Antimony			<0.020	<0.020	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.02	<0.020
Selenium			<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002	<0.002
Thallium			<0.010	<0.010	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.010
Zinc	0.09	0.09	0.015	0.013	0.04	0.071	0.095	0.061	0.03	0.046	0.05	0.022	0.028	0.028	0.013
Vanadium			<0.200	<0.200	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.2	<0.200
Silver			<0.010	<0.010	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.01	<0.010
Mercury			<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002	<0.0002
Tin			<0.020	<0.020	<0.02	<0.02	<0.02	<0.02	0.021	0.023	0.023	<0.02	0.03	0.028	<0.020
Cadmium			0.002	0.028	0.029	0.0013	0.0024	<0.001	0.0023	<0.001	0.027	0.0023	0.01	<0.001	0.025
Lead	0.0088		<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	<0.005	0.0063	0.0065	<0.005	0.0055	<0.005
TOX			<0.005	0.443	0.017	0.014	<0.005	2.64	0.393	2.29	0.4	0.154	0.125	<0.005	0.425
COX (dup)			0.000	0.336	0.024	0.013	<0.005	2.42	0.347	2.16	0.443	0.16	0.124	<0.005	0.507
COD (std. units)			<1.000	1.43	7.5	6.9	<0.2	7.6	7.7	6.8	7.6	7.1	6.8	7	6
Oil & Grease			7.6	6.7											1.39
Temperature (°C)			2	8											10
Specific Conductance (µmhos)			770	660											560
Data reported by			ERM, Inc.	ERM, Inc.	ERM, Inc. Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA

** Data not included in the calculation of average or range

A - From report dated 8 December 1981.

B: This result is of questionable qualitative significance since this constituent was detected in blanks at similar concentrations.

TABLE E-3 (continued)
TYSONS SITE
FLOODPLAIN AREA SURFACE WATER RESULTS
HSL CONSTITUENTS
mg/L

SAMPLE ID	840057** background	840071** background	FP-001	FP-002	FP-003	FP-004	FP-011**	FP-A	FP-B	FP-C	FP-D	FP-E	BA-001C
Sample Location	wet	wet					Blind	FP-004	FP-003	FP-002	FP-001	Duplicate	
Date Sampled	1/9/84	1/9/84	3/1/87	3/1/87	3/1/87	3/1/87	3/1/87	3/1/87	3/1/87	3/1/87	3/1/87	3/1/87	5/11/87
Parameter													
Aluminum	0.499	0.054		0.2 B	0.2 B	0.1 B							
Chromium	<0.010	<0.010											
Barium	0.110	0.112	0.1	0.2	0.1			0.1	0.1	0.1	0.1	0.1	0.2
Beryllium	<0.005	<0.005											
Cobalt	<0.050	<0.050											
Copper	<0.050	<0.050					0.04						
Iron	0.310	0.562	0.06	0.22	1.60	0.21			0.04		0.04		
Nickel	<0.040	<0.040											
Manganese	<0.010	0.02	0.03	0.01	0.10	0.01			0.20		0.03		1.33 J
Arsenic	<0.010	<0.010											
Antimony	<0.020	<0.020											
Selenium	<0.002	<0.002											
Thallium	<0.010	<0.010											
Zinc	0.05	<0.010			0.02 B	0.03 B		0.02 B	0.02 B		0.04 B	0.03 B	0.04 B
Vanadium	<0.200	<0.200											
Silver	<0.010	<0.010											
Mercury	<0.0002	<0.0002											
Tin	<0.020	<0.020											
Cadmium	<0.001	<0.001											
Lead	<0.005	<0.005											
TOX	<0.005	<0.005											
TOX (dup)	<0.005	0.005											
pH (std. units)	7.8	7.2											
Oil & Grease	1.2	<1.000											
Temperature (°C)	5	4											
Specific Conductance (umho)	630	405											

Data reviewed by: Beker/TSA   Beker/TSA

** Data not included in the calculation of average or range
A - From report dated 8 December 1988.
B: This result is of questionable qualitative significance since this constituent was detected in blanks at similar concentrations.

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TABLE E-3 (continued)
TYSON'S SITE
FLOODPLAIN AREA SURFACE WATER RESULTS
HSL CONSTITUENTS
mg/L

SAMPLE ID	BA-002C	BA-003C	BA-004C	BA-005C	BA-006C	BA-007C	BA-008C	BA-009	BA-010	BA-011	BA-012	BA-013	BA-014	BA-015
Sample Location														
Date Sampled	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87
Parameter														
Aluminum					0.2	0.2	0.1	0.1	0.1	0.2		0.2	0.8	
Chromium	0.2		0.1	0.3	0.1	0.2	0.1	0.2	0.4		0.2	0.2	0.1	0.01
Barium														
Beryllium														
Cobalt														
Copper														
Iron	0.97 J	0.43 J	0.33 J	0.57 J	0.18 J	4.9 J	0.25 J	0.83 J	18.4 J	0.42 J	5.15 J	1.05 J	1.5 J	1.4 J
Nickel	0.65	0.02	0.68	3.75	1.55	1.38	0.05	0.54	0.57	0.18	0.76	2.84	2.79	0.64
Manganese									0.011					
Arsenic														
Antimony														
Selenium														
Thallium														
Zinc	0.02 B	0.02 B	0.010 B	0.020 B	0.020 B	0.03 B	0.010 B	0.04 B	0.020 B	0.02 B	0.02 B	0.02	0.06 B	
Vanadium														
Silver														
Mercury														
Tin														
Cadmium														
Lead														
TOX														
TOX (sun)														
pH (std. units)														
Oil & Grease														
Temperature (°C)														
Specific Conductance (umhos)														
Data reported by														

** Data not included in the calculation of average or range

A - From report dated 8 December 1985.

B: This result is of questionable qualitative significance since this constituent was detected in blanks at similar concentration.

TABLE E-3 (Continued)
TYSON'S SITE
FLOODPLAIN AREA SURFACE WATER RESULTS
VSL ORGANIC COMPOUNDS
mg/L

Sample Number	SW007**	SW006	SW005	SW004	SW003	SW002	SW001	SW008**	SW009	840043	840046	840047	840048
Sample Location	Site A	Site B	Site C	Site D	Site E	Site F	Site G	Site H	Site I				
Date Sampled	A DKSD	A	A	A	A	A	A	A DKSD	A	1/8/84	1/8/84	1/8/84	1/8/84
Parameter													
VOLATILES													
Acetone	0.009S	0.018 0.002JS	0.078 0.058 0.092J	0.007JS	0.0084S 0.002JS	0.018	0.018 0.0079S	0.005JS	0.007JS	LT			
Methylene chloride										LT	0.0077	LT	
Toluene						0.007 0.005JS	0.005J						
Total xylenes													
2-Butanone											0.012 0.016	0.0077 0.006	
Chloroethane													
Chloroform											0.030*	0.170*	0.014*
trans-1,2-Dichloroethene													
Tetrachloroethene													
Trichloroethene													
4-Methyl-2-pentanone													
1,2,3-Trichloropropane													
Chloroethane													
Ethylbenzene													
Carbon disulfide													
SEMI-VOLATILES													
1,2,4-Trichlorobenzene													
1,2-Dichlorobenzene													
Di-n-Octyl phthalate													
Dibutyl Phthalate													
1,4-Dichlorobenzene													
Phenol													
4-Methylphenol													
Butyl benzyl phthalate													
Bis (2-ethylhexyl) phthalate													
PESTICIDES													
DDE													
DDD													
PCB-1248													
Data reported by	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA

** Data not included in the calculation of average or range
A - From report dated 9 December 1986
J: Estimated value
B: This analyte was also found in the method blank
Blank - none detected
* - or fewer
LT: Less than the reported detection limit but greater than one half of the detection limit (present but below detection limit)
All ERM data has the zero through a quality assurance review.

TABLE E-3 (Continued)
TYSON'S SITE
FLOODPLAIN AREA SURFACE WATER RESULTS
NEL ORGANIC COMPOUNDS
mg/L

Sample Number	840051	840053	840055	840057	840059	840061	840063	840065	840067	840069	840071	840073	840047
Sample Location										Background			
Date Sampled	1/9/84	1/9/84	1/9/84	1/10/84	1/10/84	1/10/84	1/9/84	1/10/84	1/10/84	1/10/84	1/10/84	1/10/84	1/10/84
Parameter													
VOLATILES													
Acetone		0.13											
Methylene chloride	LT	LT	LT	LT	LT	LT	LT	LT		LT		LT	
Toluene		0.0083	0.06			0.045	0.0066		0.006				
Total Xylenes			0.10			0.12			0.0007				
2-Butanone													
Chloroform			0.006					0.007					
Chloroethane													LT
trans-1,2-Dichloroethene								LT					
Trichloroethene		LT	0.011			0.0084	LT	LT					0.0077
Trichloroethane		LT	0.01			0.0006		LT					0.006
4-Methyl-2-pentanone		LT	LT										
1,2,3-Trichloropropane			1.700*		0.330*	0.918*	0.820*	0.064*	0.140*	0.007*			0.170*
Chlorobenzene	0.024	0.021											
Ethylbenzene						0.029							
Carbon disulfide					0.042	0.053							
SEMI-VOLATILES													
1,2,4-Trichlorobenzene		LT											
1,2-Dichlorobenzene		LT		LT						LT			
Di-n-Octyl phthalate													
Dimethyl Phthalate													
1,4-Dichlorobenzene													
Phenol													
4-Nitrophenol													
Butyl benzyl phthalate													
bis-(2-ethylhexyl) phthalate													
PESTICIDES													
DDE	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA	Below/TSA
DDD													
PCB-1248													

Data reported by: Below/TSA

** - Does not included in the calculation of average or range
A - First report dated 8 December 1983
J: Estimated value
B: This sample was also found in the method blank
Blank - none detected
~ - or lower
LT: Less than the method detection limit but greater than one half of the detection limit (present but below detection limit)
All ERM data are greater through a quantity assurance review.

TABLE E-3 (Continued)
TYSON'S SITE
FLOODPLAIN AREA SURFACE WATER RESULTS
H&L ORGANIC COMPOUNDS
mg/L

Sample Number	840057 ** Background	840071 ** Background	FP-001	FP-002	FP-003	FP-004	FP-011	BA-001C	BA-002C	BA-003C	BA-004C	BA-005C	BA-006C
Sample Location Date Sampled	1/18/84	1/18/84	3/87	3/87	3/87	3/87	3/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87	5/11/87
Parameter													
VOLATILES													
Acetone	LT					0.006 B			0.003 NV		0.03 NV	0.7 NV	0.004 NV
Methylene chloride													
Toluene									0.011				
Test xylene													
2-Butanone													
Chloroform													
Chloroform								0.003 B		0.003 B	0.002 B		
trans-1,2-Dichloroethene													
Tetrachloroethene													
Trichloroethene			0.006										
4-Methyl-2-pentanone			0.128	0.002 B	0.006	0.013		0.012	0.073	0.020	0.020		
1,2,3-Trichloropropane													
Chloroethane													
Ethylbenzene													
Carbon disulfide													
SEMI-VOLATILES													
1,2,4-Trichlorobenzene													
1,2-Dichlorobenzene									0.006 J				
Di-n-Octyl phthalate													
Diethyl Phthalate									0.006 J				
1,4-Dichlorobenzene													
Phenol													
4-Methylphenol													
Butyl benzyl phthalate										0.006 B			
Bis (2-ethylhexyl) phthalate													
PESTICIDES													
DDE										0.00006 J			
DDD										0.0001			
PCB-1248													
Data reported by													

** Does not included in the calculation of average or range
A - From report dated 8 December 1988
J - Estimated value
B: This analyte was also found in the method blank
Blank = not detected
* or lower
LT: Less than the specified detection limit but greater than one half of the detection limit (present but below detection limit)
All ERM data from grab through a quality assurance review.

AR301556

TABLE 6-3 (Continued)
TYSON'S SITE
FLOODPLAIN AREA SURFACE WATER RESULTS
MISL ORGANIC COMPOUNDS
mg/L

Sample Number	BA-007C	BA-008C	BA-009	BA-010	BA-011	BA-012	BA-013	BA-014	BA-015	1479 Stripper Effluent	1481 Stripper Effluent Duplicate	1478 Stripper Influent	1481 Stripper Effluent Duplicate	1452 Weir 4
Date Sampled	8/11/87	8/11/87	8/11/87	8/11/87	8/11/87	8/11/87	8/11/87	8/11/87	8/11/87	8/17/87	8/17/87	8/17/87	8/17/87	8/18/87
Parameter														
VOLATILES														
Acetone	0.23 NV	0.095 NV		0.07 NV	0.096 NV	0.09 NV	0.004 NV	0.004 NV						
Methylene chloride				0.009										
Toluene	0.004 J			0.052										
Total xylenes														
2-Butanone														
Chloroform	0.002 B			0.002 J										
Chloroethane														
trans-1,2-Dichloroethene				0.002 J								0.0012 J		
Tetrachloroethene				0.004 J										
Trichloroethene			0.006		0.018	0.006				0.16	0.15	0.17	0.15	0.014
4-Methyl-2-pentanone				0.002 J										
1,2,3-Trichloropropane														
Chloromethane														
Ethylbenzene														
Carbon disulfide														0.0054
SEMI-VOLATILES														
1,2,4-Trichlorobenzene				0.007 J						XX	XX	XX	XX	
1,2-Dichlorobenzene										XX	XX	XX	XX	
Di-n-Octyl phthalate				0.018						XX	XX	XX	XX	
Dimethyl Phthalate				0.006 J						XX	XX	XX	XX	
1,4-Dichlorobenzene				0.104						XX	XX	XX	XX	
Phenol										XX	XX	XX	XX	
4-Methylphenol								0.01 B		XX	XX	XX	XX	
Butyl benzyl phthalate				0.008 B						XX	XX	XX	XX	
Bis (2-ethylhexyl) phthalate										XX	XX	XX	XX	
PESTICIDES														
DDE					0.0002				0.0002					
DDD					0.0035									
PCB-1248														

Data reported by

** Data not included in the calculation of average or range
A - From report dated 8 December 1988
J - Estimated value
B - This analyte was also found in the method blank
NV - Blank = none detected
" - or lower
LT: Less than the specified detection limit but greater than one half of the detection limit (present but below detection limit)
All ERM data too poor through z quality assurance review.

TABLE E-4
TYSON'S SITE
FLOODPLAIN AREA SOIL RESULTS
HSL INORGANIC CONSTITUENTS
mg/kg, dry weight

Sample ID	W of Ice House (Background) SS-066*	Western Swamp Area SS-067*	Western Swamp Area SS-068**	Air Stripper Outlet SS-069**	Air Stripper Outlet SS-070	840034	840035	840036	840037
Date Sampled	A	A	A	A	A	1/8/84	1/8/84	1/8/84	1/8/84
Parameter									
Aluminum	16800	9270	15400	14400	9200	8920	10100	5950	6700
Antimony						<1	<1	<1	<1
Arsenic	8.5	26	14.8	16	11	13	10	6.0	9.4
Barium	90	240	149	640	245	92	82	110	94
Beryllium	0.7	0.8	0.74	3	1.02	0.61	0.69	0.54	0.54
Cadmium	0.18	0.8	0.58	0.96	0.25	0.78	1.3	0.78	1.0
Chromium	31	30	22.3	40	18.4	12	14	6.8	9.3
Cobalt	10	20	12.4	9	8.2	6.7	10	3.3	4.9
Copper	30	450	108	110	34.8	25	13	30	15
Iron	28800	30900	14800	25900	11900	12900	14000	7160	10800
Lead	66	190	124	552	55.2	66	60	61	59
Manganese	492	940	344	211	129	252	566	235	59
Mercury	0.13NV	0.9NV	0.9NV	0.39NV	0.41NV	<0.1	<0.1	<0.1	<0.1
Nickel	16	20	17.3	23	12.3	9.7	12	6.0	6.6
Selenium	0.78	28	2	2.3J	1.2	1.2	<1	1.1	<1
Silver	0.12NV	0.25NV		0.45		<0.5	<0.5	<0.5	<0.5
Thallium						<0.5	<0.5	<0.5	<0.5
Tin	108	405	37.1	205	24.5	18	21	13	13
Vanadium	38	405		54		18	16	11	18
Zinc	112	127	205	3070	243	57	57	41	51
% Moisture	32.1	75.5							
pH	6.47	6.17		5.91					
TOX						<100	<100	<100	<100
TOC									
Data reported by	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	Beker/TSA	Beker/TSA	Beker/TSA	Beker/TSA

A - Data taken from 8 December 1988 report
B - this analyte was also found in the method blank and is of questionable qualitative significance
NV - this result is not valid; the laboratory absorbance data indicated this concentration is below the detection capability
All ERM data has gone through a quality assurance review.

J - estimated value
Blanks indicate not detected
* - Large volume composite
** - Grab samples to obtain preliminary data

Sample ID Date Sampled Parameter	840008 1/8/84	FP-001 3/ /87	FP-002 3/ /87	FP-003 3/ /87	FP-004 3/ /87	FP-005 3/ /87	FP-006 3/ /87	FP-007 3/ /87	FP-008 3/ /87	FP-009 3/ /87
Aluminum	5240	4410.	11000.	17000.	8090.	N/A	N/A	N/A	N/A	N/A
Antimony	<1					N/A	N/A	N/A	N/A	N/A
Arsenic	7.4	4.93	5.99	9.08	4.72	N/A	N/A	N/A	N/A	N/A
Barium	71	82.7	93.8	108	91.3	N/A	N/A	N/A	N/A	N/A
Beryllium	0.42	0.29	0.54	0.96	0.63	N/A	N/A	N/A	N/A	N/A
Cadmium	0.72		0.41 B	2.23	0.79	N/A	N/A	N/A	N/A	N/A
Chromium	7.5	6.7	19.0	57.3	20.5	N/A	N/A	N/A	N/A	N/A
Cobalt	3.0	4.4	12.2	31.8	11.0	N/A	N/A	N/A	N/A	N/A
Copper	16	23.2	28.5	78.8	40.9	N/A	N/A	N/A	N/A	N/A
Iron	10900.	11600.	16300	28900	13500	N/A	N/A	N/A	N/A	N/A
Lead	47	47.9	72.0	104	55.1	N/A	N/A	N/A	N/A	N/A
Manganese	33	437.	457	1470	466.	N/A	N/A	N/A	N/A	N/A
Mercury	<0.1					N/A	N/A	N/A	N/A	N/A
Nickel	4.8	7.3	16.3	49.4	17.3	N/A	N/A	N/A	N/A	N/A
Selenium	<1		1.36	2.07	0.94	N/A	N/A	N/A	N/A	N/A
Silver	<0.5			0.48		N/A	N/A	N/A	N/A	N/A
Thallium	<0.5									
Tin	6.0									
Vanadium	14	4.4	25.8	38.2	14.2	N/A	N/A	N/A	N/A	N/A
Zinc	50	61.1	115	201	156.	N/A	N/A	N/A	N/A	N/A
% Moisture										
pH										
TOX										
TOC	N/A	12,000	23,000	24,000	13,000	6,500	9,600	17,000	6,500	15,000
Data reported by	Dalen/TSA									

A - Data values from 8 December 1986 report
B - this analyte was also found in the method blank and is of questionable qualitative significance
NV - this result is not valid; the laboratory absorbance data indicated this concentration is below the detection capability
All EPA data has gone through a quality assurance review,

J - estimated value
Blanks indicate not detected
* - Large volume composite
** - Grab samples to obtain preliminary data

TABLE E-4
TYSONS SITE
HILLSIDE AREA SOIL RESULTS
VAL INORGANIC CONSTITUENTS
mg/kg, dry weight basis

Footnotes:
A - Data taken from 8 December 1986 report
B - this analyte was also found in the method blank and is of questionable qualitative significance
J - estimated value
(Blank) indicates not detected
** - Data not included in the calculation of average or range
NV - this result is not valid; the laboratory detection data indicated the concentration is below the detection capability
NDB - Not detected due to laboratory blank correction
NV - Not detected per Appendix D of Baker/TSA Onsite RI Report
All ERM data has gone through a quality assurance review

TABLE E-6
TYSON'S SITE
SEEP AREA SOIL RESULTS
HSL INORGANIC CONSTITUENTS
mg/kg, dry weight basis

Sample Number	SS 001	SS 002	SS 003	SS 004	SS 005	SS 006	SS 007	SS 008	SS 009	SS 010	SS 011
Sample Date	A	A	A	A	A	A	A	A	A	A	A
Parameter											
Aluminum	8770	12900	9160	7700	9720	9390	11100	9500	10500	9710	9420
Antimony											
Arsenic	4.31	2.84B	5.8	3.50B	5.43	3.84B	4.44	2.61B	4.72	3.99	2.99
Barium	74	138	196	58	98	38	96	126	104	117	90
Beryllium	0.37	0.48	0.58	0.47	0.37	0.24	0.358	0.57	0.55	0.35	0.1INV
Cadmium							0.12				0.1
Chromium	9.9	13.8	22.8	8.2	14.8	10.8	14.8	5.7	13.8	10.8	12
Cobalt	4.9	4.8	4.8	3.5	4.9	4.6	4.9	2.3	3.5	3.5	3
Copper	6.2	5.7	8.3	4.7	9.9	4.6	6.8	3.4	8.1	8.2	9
Iron	7300	12200	21400	8210	9810	7090	10900	4250	8780	7910	9050
Lead	18.5	5.7	8.5	14	23.4	10.8	18.8	4.98	18.4	18.8	23
Manganese	151	172	122	108	223	116	146	102	320	317	125
Mercury							0.128				0.08NV
Nickel	7.4	10.3	7.1	7	6.2	6	7.4	4.6	5.8	4.7	7
Selenium											0.38
Silver											0.08NV
Thallium											
Tin											0.28
Vanadium	13.5	12.8	13.1	11.7	16	13.1	17.3	9.8	17.3	14.1	12
Zinc	26.7	11.8	21	20.8	36.7	21.1	31.2	7.71	34	49.8	78.8
pH	6.42	6.62	6.58	5.98	7.03	7.20	7.01	8.24	7.38	7.48	7.03
TOC											
Data reported by	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc

A - Data taken from 8 December 1986 report
IV - not valid
J - estimated value
B - this result is of questionable qualitative significance since the compound was detected in the blank
INV - invalid data
NDB - not detected but in blank
Bold indicates compound not detected
** Data not included in the calculation of average or range
All EPA data has gone through a quality assurance review.

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TABLE E-9
TYSON'S SITE
SEEP AREA SOIL RESULTS
HSL INORGANIC CONSTITUENTS
mg/kg, dry weight basis

Sample Number	SS 012	SS 013	SS 014	SS 015	SS 016	840018	840020	840152	840153	840154 duplicate	840032 background
Sample Date	A	A	A	A	A	1/7/84	1/7/84	2/28/84	2/28/84	2/28/84	1/8/84
Parameter											
Aluminum	15000	15500	11000	13000	13000	8000	3450	4990	6970	7900	10100
Antimony						<1	<1	<1	<1	<1	<1
Arsenic	14.3	9	8.59	7.28	4.63	7.8	4	3.3	4.8	3.8	14
Barium	95	84	58	141	34	47	37	88	48	50	204
Beryllium	0.83	0.44	0.278	0.47	0.238	0.38	<0.25	0.5	0.25	0.5	0.8
Cadmium		0.13				3.7	3	0.1	<0.05	<0.05	0.77
Chromium	55.8	27.8	18	24.7	16.9	5.5	2	4.5	5	6	8.8
Cobalt	15.4	7.2	2.2	5.8	4.5	3.4	<2.5	2.5	2.5	5	4.5
Copper	34.3	22.5	4.5	11.8	4.5	100	<2.5	5	2.5	5	10
Iron	34100	18000	15700	21000	12200	8150	2820	2960	3000	4700	9500
Lead	18.9	29.7	7.9	9.4	7.8	23	9.3	29	8.7	8.8	8.3
Manganese	512	331	98.4	198	115	131	39	121	97	101	900
Mercury		0.06NV				<0.1	<0.1	NV	NV	NV	<0.1
Nickel	27.2	13.2	4.5	8.2	7.9	8.5	5.5	4	8	8	7.9
Selenium		0.75				<1	<1	<1	<1	<1	<1
Silver		0.2NV				<0.5	<0.5	0.2	<0.1	<0.1	<0.5
Thallium						<0.5	<0.5	<0.5	<0.5	<0.5	<0.5
Tin						15	7.5	NDB	NDB	NDB	25
Vanadium	58.2	25.8	22.5	27.1	20.3	<10	<10	<10	<10	<10	17
Zinc	79.2	77.2	19	33.1	19.1	19	4	24.5	14	15.5	55
pH	7.45	7.33	5.04	6.57	4.96						
TOC						<100	<100	<100	<100	<100	<100
Data reported by	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	EPA, Inc	Beter/TSA	Beter/TSA	Beter/TSA	Beter/TSA	Beter/TSA	Beter/TSA

A - Data taken from 8 December 1986 report
NV - not valid
J - estimated value
B - J's result is of questionable qualitative significance since the compound was detected in the blank
INV - invalid data
NDB - not detected due to blanks
Blanks indicate compound not detected
** - Data not included in the calculation of average or range
All EPA data has gone through a quality assurance review.

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TABLE E-10.
TYSON'S SITE
RAILROAD AREA SEDIMENT RESULTS
HSL INORGANIC CONSTITUENTS
mg/kg, dry weight basis

Sample Number	840044	840048	840058** Background west	840181** Background west	840072** Background east
Date Sampled	1/9/84	1/9/84	1/9/84	2/28/84	1/10/84
Parameter					
Aluminum	1840	1830	1920	2580	2760
Chromium	2.1	9.7	2.1	2.5	2.8
Barium	34	64	17	46	48
Beryllium	<0.25	0.48	<0.25	<0.25	0.35
Cobalt	<2.5	3.6	<2.5	<2.5	<2.5
Copper	<2.5	17	<2.5	8	3.1
Iron	2410	8340	1830	2630	3870
Nickel	<2	6	2.6	2	2.6
Manganese	94	185	70	93.8	149
Arsenic	3.1	5.4	3.8	1.7	4.3
Antimony	<1	<1	<1	<1	<1
Selenium	<1	<1	<1	0.2	<1
Thallium	<0.5	<0.5	<0.5	<0.5	<0.5
Zinc	11	3.5	24	52	14
Vanadium	<10	<10	<10	<10	<10
Silver	<0.5	<0.5	<0.5	<0.5	<0.5
Mercury	<0.1	<0.1	<0.1	INV	<0.1
Tin	3.5	<1	5.3	NDB	5.4
Cadmium	0.47	0.87	<0.05	0.1	0.26
Lead	9.5	76	11	17	18
TOX	<100	<100	<100	<100	<100
Oil & Grease	<100	200	<100		<100
Data reported by	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA

** Data not included in the calculation of average or range
Blank indicate compound not detected
INV - Invalid data
NDB - Not detected due to blank
All ERM data has gone through a quality assurance review.

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TABLE E-10
TYSON'S SITE
RAILROAD AREA SEDIMENT RESULTS
HSL ORGANIC COMPOUNDS
mg/kg, dry weight basis

Sample I.D.	840044	840046	840058 ** background	840151 ** background	840072 ** background
Date Sampled	1/9/84	1/9/84	1/9/84	2/28/84	1/10/84
Parameter					
VOLATILES					
Chlorobenzene				INV	
Chloroform				INV	
1,2-Dichloropropane				INV	
Ethylbenzene				INV	
Methylene chloride	0.012	0.028	0.006	INV	LT
Fluorotrichloromethane	0.0049		0.004	INV	LT
Tetrachloroethene		0.028		INV	
Toluene				INV	
Trichloroethene		0.0094		INV	
o-Xylene				INV	
1,2,3-Trichloropropane		0.036			
SEMI-VOLATILES					
Phenol				INV	
Benzoic Acid		10		INV	
Cresol*				INV	
Acenaphthene					LT
Fluoranthene		1.6	1.4		
Bis (2-ethylhexyl) phthalate		LT		0.53	
Di-n-Butyl phthalate		LT		2.3	
Benzo(a)anthracene		0.6	0.72		
Benzo(a)pyrene		LT	LT		
Benzo(b&k)fluoranthene		1.3	LT		
Chrysene		0.9	0.76	1.2	
Anthracene		LT	LT		
Benzo(ghi)perylene		1.0	LT		
Fluorene				3.3	
Phenanthrene		1.1	0.72	2.1	
Dibenzo(a,h)anthracene		1.5			
Indeno(1,2,3-cd)pyrene		1.0	LT		
Pyrene		1.2	1.2	2.3	
Dibenzofuran		LT			
PESTICIDES					
4,4'-DDD				1.100PN	
Beta-BHC				0.055PN	
PCB-1254			0.250PN	5.200PC	
PCB-1260		0.110PN			
Data prepared by	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA	Baker/TSA

INV - Invalid data
Cresol* - 2- and 4-methyl phenol.
PN - pesticide or PCB not confirmed by GC/MS.
LT - less than the specified detection limit but greater than one half of the detection
    limit (present but below the detection limit)
PC - pesticide or PCB confirmed by GC/MS.
All ERM data has gone through a quality assurance review.
** Data not included in the calculation of average or range

APPENDIX F

FLOODPLAIN/WETLAND AREA
(OPERABLE UNIT 5)
BIOLOGICAL/ENVIRONMENTAL
ASSESSMENTS

APPENDIX F

(Taken from Subsection 4.6.4 Off-Site Operable Unit RI)

4.6.4 Biological Studies

4.6.4.1 Environmental Mobility of Organic Chemicals

The results of the environmental mobility analysis for organic chemicals in the Floodplain/Wetlands Operable Unit (Section 3.5.3) are given in Table 4-28. The organic compounds are divided based upon their K_{OC} values into three categories: no, low to moderate, and high bioaccumulation potential.

Little or no bioaccumulation is predicted for six compounds in Table 4-28, based upon the K_{OC} values. These compounds prefer the aquatic and atmospheric media to soils or sediments. Thus, these compounds could migrate through the soil column to the ground water or runoff to surface waters. The low to moderate bioaccumulators have some sorption to soils and sediments, but most would preferentially be found in aquatic and atmospheric media. The compounds might sorb to the soils and/or bioaccumulate to varying degrees in aquatic and terrestrial animals. The third category, compounds with high bioaccumulation potential, is represented by compounds which tend to sorb to soils/sediments in preference to water and air. These compounds do not easily leach or volatilize and thus, are more readily

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TABLE 4-28
BIOACCUMULATION PROTENTIAL FOR
ORGANIC COMPOUNDS DETECTED DURING THE EPA ON-SITE AND ERM FLOODPLAIN INVESTIGATIONS

COMPOUND	Kow	Koc	RANKING FOR BIOACCUMULATION
NO BIOACCUMULATION POTENTIAL			
ENDOSULFAN I	2.00E-02	8.60E-03	77
ENDOSULFAN II	2.00E-02	8.60E-03	76
ACETONE	5.75E-01	2.20E+00	75
2-BUTANONE (TID)	2.60E-01	4.50E+00	74
VINYL CHLORIDE	1.70E+01	8.20E+00	73
METHYLENE CHLORIDE	1.82E+01	8.50E+00	72
LOW TO MODERATE BIOACCUMULATION POTENTIAL			
ANILINE		1.40E+01	71
PHENOL	3.00E+01	1.42E+01	70
2-METHYLPHENOL	9.33E+01	1.48E+01	69
4-METHYLPHENOL	9.33E+01	1.48E+01	68
N-NITROSODI-N-PROPYLAMINE	3.10E+01	1.50E+01	67
4-METHYL-2-PENTANONE		1.99E+01	66
2-HEXANONE		1.99E+01	65
1,1-DICHLOROETHANE	6.30E+01	3.00E+01	64
NITROBENZENE	7.40E+01	3.60E+01	63
CHLOROFORM	9.10E+01	4.40E+01	62
TRANS-1,3-DICHLOROPROPENE	1.00E+02	4.50E+01	61
CIS-1,3-DICHLOROPROPENE	1.00E+02	4.50E+01	60
1,2-DICHLOROPROPANE	1.05E+02	5.10E+01	59
CARBON DISULFIDE (TID)	2.00E+00	5.40E+01	58
BENZOIC ACID (TID)	7.40E+01	5.55E+01	57
TRANS-1,2-DICHLOROETHENE	1.23E+02	5.90E+01	56
BENZENE	1.35E+02	6.50E+01	55
1,2,3-TRICHLOROPROPANE		6.95E+01	54
2-CHLOROPHENOL	1.51E+02	7.30E+01	53
ISOPHORONE	1.50E+02	8.70E+01	52
2,4-DIMETHYLPHENOL	2.00E+02	9.60E+01	51
1,1,2,2-TETRACHLOROETHANE	2.45E+02	1.16E+02	50
TRICHLOROETHENE	2.63E+02	1.26E+02	49
DIETHYL PHTHALATE	2.95E+02	1.42E+02	48
1,1,1-TRICHLOROETHANE	3.20E+02	1.52E+02	47
FLUOROTRICHLOROMETHANE	3.31E+02	1.59E+02	46
HEPTACHLOR EPOXIDE	4.50E+02	2.20E+02	45
O-XYLENE	1.62E+03	2.40E+02	44
TOLUENE	6.20E+02	3.00E+02	43
CHLOROBENZENE	6.90E+02	3.30E+02	42
TETRACHLOROETHENE	7.59E+02	3.84E+02	41
N-NITROSODIPHENYLAMINE	1.35E+03	6.45E+02	40
2-METHYLNAPHTHALENE		7.12E+02	39
NAPHTHALENE	1.95E+03	8.40E+02	38
HIGH BIOACCUMULATION POTENTIAL			
ETHYLBENZENE	2.20E+03	1.10E+03	37
DIELDRIN	3.50E+03	1.70E+03	36
1,2-DICHLOROBENZENE	3.60E+03	1.70E+03	35
1,3-DICHLOROBENZENE	3.60E+03	1.70E+03	34
1,4-DICHLOROBENZENE	3.60E+03	1.70E+03	33
ACENAPHTHYLENE	5.10E+03	2.50E+03	32
BETA-BHC	7.80E+03	3.80E+03	31
ALPHA-BHC	7.80E+03	3.80E+03	30
GAMMA-BHC	7.80E+03	3.80E+03	29
ACENAPHTHENE	8.80E+03	4.60E+03	28
2-CHLORONAPHTHALENE	1.00E+04	4.50E+03	27
DELTA BHC	1.40E+04	6.50E+03	26
FLUORENE	1.50E+04	7.30E+03	25
1,2,4-TRICHLOROBENZENE	1.90E+04	9.20E+03	24
HEPTACHLOR	2.60E+04	1.20E+04	23
PHENANTHRENE	2.60E+04	1.40E+04	22

TABLE 4-25 (continued)
BIOACCUMULATION PROTENTIAL FOR
ORGANIC COMPOUNDS DETECTED DURING THE EPA ON-SITE AND ERM FLOODPLAIN INVESTIGATIONS.

COMPOUND	Kow	Koc	RANKING FOR BIOACCUMULATION
ANTHRACENE	2.80E+04	1.40E+04	21
FLUORANTHENE	7.90E+04	3.80E+04	20
PYRENE	8.00E+04	3.80E+04	19
ALDRIN	2.00E-05	9.60E+04	18
CHLORDANE	3.00E+05	1.40E+05	17
DI-N-BUTYL PHTHALATE	3.50E+05	1.70E+05	16
BUTYL BENZYL PHTHALATE	3.50E+05	1.70E+05	15
CHRYSENE	4.10E+05	2.00E+05	14
BENZO(A)ANTHRACENE	4.10E+05	2.10E+05	13
BENZO(K)FLUORANTHENE	1.15E+06	5.50E+05	12
BENZO(B)FLUORANTHENE	1.15E+06	5.50E+05	11
4,4'-DDD	1.60E+06	7.70E+05	10
INDENO(1,2,3-CD)PYRENE	3.20E+06	1.60E+06	9
BENZO(GHI)PERYLENE	3.20E+06	1.60E+06	8
DIBENZO(A,H)ANTHRACENE	6.90E+06	3.30E+06	7
4,4'-DDT	8.10E+06	3.90E+06	6
4,4'-DDE	8.10E+06	4.40E+06	5
BENZO(A)PYRENE	1.15E+06	5.50E+06	4
PCB	1.40E+07	6.70E+06	3
BIS(2-ETHYLHEXYL)PHTHALATE	4.10E+08	2.00E+09	2
DI-N-OCTYL PHTHALATE	7.40E+09	3.60E+09	1

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available for bioaccumulation in fish and/or mammals. The PAHs,
pesticides/PCBs, and phthalate esters are commonly detected in
tissues of exposed fish or animals. These compounds have the
potential to biomagnify in the food chain.

On this basis, analyses for the turtle fat and tissue, clam
samples, and plant samples from the floodplain included Hazardous
Substance List (HSL) volatiles, semi-volatiles (both base
neutrals and acid extractables), pesticides/PCBs, and
1,2,3-trichloropropane. The HSL covers the compounds from Table
4-19 and additional compounds which might be potential
bioaccumulators.

4.6.4.2 Bioaccumulation Studies

Table 4-29 is a summary of the HSL organic compounds detected in
the turtle, clam, and plant samples collected for the
bioaccumulation study. Inspection of Table 4-29 and the Quality
Assurance report for these samples shows that many problems were
encountered during the analysis of the samples which subsequently
makes data interpretation difficult. Most of the problems are a
result of the methods employed for sample storage, preparation,
concentration, and analysis. At the time of this work the EPA
methods for analyzing biological samples had not been fully
developed. The analytical results of several biological samples
suffered from serious matrix effects which required the reporting
of high detection limits and questionable results by the
laboratory. After an ERM data review it was deemed necessary to
qualify all of the data reported with the exception of the
PCB-1260 concentrations in the turtle fat tissue samples. Also,

TABLE 4-29

COMPOUNDS DETECTED IN BIOLOGICAL SAMPLES, TYSON'S SITE
OFF-SITE OPERABLE UNIT RI

Units ug/kg

Compounds	Turtle				Clam		Imperions		
	Muscle-C	Muscle-S	Fat-C	Fat-S	Clam-C	Clam-BP	Sump	A.S.	C
methylene chloride	15 B	33 B	24 B	69 B	34 B	37 B	31 B	27 B	23 B
acetone	840 NY	1500 NY	1200 NY	3100 NY	330 NY	7500 NY	990 NY	790 NY	710 NY
carbon disulfide	10 J	11 J	ND	ND	ND	ND	ND	ND	ND
chloroform	11 J	21 J	ND	600 J	90 J	26 J	16 J	44 J	55 J
2-butanone	8 B	14 B	ND	ND	10 B	ND	13 B	23 B	9 B
benzene	14 J	37 J	ND	1500 J	100 J	23 J	8 J	84 J	41 J
toluene	3 B	3 B	ND	34 J	5 B	8 B	ND	5 B	ND
bis (2-ethylhexyl) phthalate	290 H	ND	ND	ND	120 H	50 H	360 H	ND	140 H
benzoic acid	ND	ND	ND	ND	290 J	200 J	ND	ND	ND
benzyl alcohol	ND	ND	ND	ND	ND	ND	410 J	1000 J	370 J
beta-BHC	ND	ND	ND	ND	ND	ND	18 NR	ND	ND
PCB-1260	ND	ND	69,000	19,000	ND	ND	ND	ND	ND
% Lipids	0.40	0.65	71.9	77.2	1.13	1.13	0.51	0.29	0.30

S - site sample   AS - airstripper   ND - not detected   C - control sample   BP - Bridgeport

Qualifier Codes

B - Result is questionable qualitative significance, compound was detected in laboratory blanks at similar concentrations.

J - Result considered a quantitative estimate - refer to quality assurance review.

NY - Results for acetone are invalid - acetone was used for decontamination of equipment.

NR - Result is not reliable - refer to quality assurance review.

H - Result is suspected unreliable since this compound is frequent lab contaminant - refer to quality assurance review for additional details.

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as discussed below, the presence or absence of particular compounds in the biological samples does not necessarily reflect their environmental occurrence or source.

Both the control(C) or upgradient turtle and downgradient or site turtle(S) had elevated PCB-1260 concentrations in the fat samples; 69,000 and 19,000 ug/kg, respectively. The source(s) of the PCB-1260 are unknown. The only confirmed detections of PCBs were noted in upgradient background samples (250 and 5200 ppb in soil) collected during the On-Site RI conducted by Baker. Much lower (less than 100 ppb) but unconfirmed concentrations of PCBs were reported in off-site samples also collected during the Baker investigation. One of the leachates generated for the additional sediment toxicity bioassays, discussed in Section 4.6.4.4, reported a PCB 1248 concentration of 3.5 ug/l. No quantifiable concentrations of PCBs were reported in any of the samples collected in the former lagoon area. However, it may be noted that trace levels of PCBs which themselves may not be detected/reported by a laboratory in environmental samples can be bioaccumulated to the levels which have been detected in the turtle fat sample.

Although a detailed pathologic examination could not be conducted on the frozen turtle specimens, the turtles were inspected for gross abnormalities. Neither turtle showed evidence of gross abnormalities (Appendix O).

No detectable concentrations of 1,2,3-trichloropropane; the predominant site contaminant and major component of the dense nonaqueous phase liquid present in the deep aquifer, were reported in any of the biological samples. The detection limit

for the 1,2,3-trichloropropane was reported to be 5 ppb for these samples. However, as stated in the ERM data review, the extended period of sample storage prior to analysis may have resulted in losses of VOA analytes including 1,2,3-trichloropropane from the sample.

Because of excessive sample holding time by the laboratory, the reported results for volatile organic compounds (VOCs) cannot be regarded with any measure of confidence. The VOCs reportedly detected in tissue samples are common laboratory solvents and can easily adulterate samples stored for long periods. It may be noted in this context that no detections of trichloropropane, which is not a common laboratory contaminant, were reported in these samples.

According to the Superfund Public Health Evaluation Manual (1986), the bioconcentration factors for VOCs are relatively low (benzene and chloroform are 5.2 and 3.75, respectively). Biomagnification of these compounds is of minimal significance.

It should also be noted that none of the major site-related HSL organic compounds (primarily polynuclear aromatics (PAHs)) were detected in a quantifiable concentration above the reported detection limits in any of the biological samples. PNAs can be efficiently metabolized by the liver to more polar compounds which are conjagated and rendered even more water soluble and are readily excreted. Some literature indicates that this metabolic activity is limited in shellfish. The absence of detectable PNAs in clam tissues may suggest that transport of these contaminants to sensitive habitats is minimal.

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4.6.4.3 Large Volume Acute and Chronic Bioassays

Soil samples were obtained from 3 locations and submitted to the Academy of Natural Sciences in Philadelphia for generation of leachate for use in the acute and chronic bioassays. Soils were sampled on two separate occasions for subsequent chemical analyses. On 28 July 1986, samples were collected at three locations (Plate 6). These were:

- 1000 feet west of the ice-house (off site-control)

- western swamp (area near are railroad signal tower)

- air stripper outfall (immediate vicinity of the discharge pipe)

On 25 September 1986, a second set of samples was collected in the west swamp and air stripper outfall. Analytical results for both inorganic constituents and organic compounds are presented in Table 4-30 and discussed below.

Organic Compounds

The ice-house sample, which was collected approximately 2000 feet west of the Floodplain/Wetlands Operable Unit, contained a number of PAH compounds (excluding the estimated values) including: benzo(a)anthracene, benzo(a)pyrene, benzo(b)fluoranthene, benzo(k)fluoranthene, chrysene, fluoranthene, phenanthrene, and pyrene. Excluding the estimated PAH concentrations, PAHs were not found in the air stripper outfall samples. Pyrene and Indeno

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TABLE   4-30
TYSON'S SITE
FLOODPLAIN AREA SOIL RESULTS
HSL INORGANIC CONSTITUENTS
mg/kg, dry weight

Sample ID Date Sampled Parameter	W of Ice House (Background) SS-066* A	Western Swamp Area SS-067* A	Western Swamp Area SS-068* A	Air Stripper Outfall SS-069** A	Air Stripper Outfall SS-070 A
Aluminum	16600	9270	15400	14400	9200
Antimony					
Arsenic	8.5	26	14.9	16	11
Barium	90	240	148	860	245
Beryllium	0.7	0.9	0.74	3	1.02
Cadmium	0.18	0.8	0.55	0.96	0.28
Chromium	31	30	22.3	40	19.4
Cobalt	16	20	12.4	9	8.2
Copper	30	450	108	110	34.9
Iron	26600	30900	14600	25600	11600
Lead	68	180	194	652	55.2
Manganese	462	940	344	211	129
Mercury	0.13NV	0.6NV	0.6NV	0.36NV	0.41NV
Nickel	16	20	17.3	23	12.3
Selenium	0.78	28	2	2.3J	1.2
Silver	0.12NV	0.25NV		0.48	
Thallium					
Tin	108	408		208	
Vanadium	38	408	37.1	54	24.5
Zinc	112	127	205	3070	243
% Moisture	32.1	75.5			
pH	6.47	6.17		5.91	
TOX					
Data reported by	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.

A - Data taken from 8 December 1986 report

* - Large volume composite

** - Grab samples to obtain preliminary data

B - this analyte was also found in the method blank and is of questionable qualitative significance

J - estimated value

NV - this result is not valid; the laboratory absorbance data indicated this concentration is below the detection capability

Blanks indicate not detected

All ERM data has gone through a quality assurance review.

TABLE   4-30 (Continued).
TYSON'S SITE
FLOODPLAIN AREA SOIL RESULTS
HSL INORGANIC CONSTITUENTS
mg/kg, dry weight

Sample ID	W of Ice House (Background) SS-066*	Western Swamp Area SS-067*	Western Swamp Area SS-068*	Air Stripper Outfall SS-069**	Air Stripper Outfall SS-070
Date Sampled	A	A	A	A	A
Parameter					
VOLATILES					
1,2,3-Trichloropropane				8.3	0.033
Methylene chloride	0.063B	0.115	0.064B	0.138	0.067B
Acetone	0.13B	0.24B	0.46B	0.27B	0.33B
Chloroform	0.009B				
Vinyl chloride					
1,1-Dichloroethane		0.020J			
trans-1,2-Dichloroethene		0.040J			
2-Butanone		0.040B	0.099B	0.04B	0.041B
Trichloroethene				0.04	
Tetrachloroethene				0.06	
Toluene		0.22J	0.02B	0.02B	
Chlorobenzene		0.25J	0.045J	0.09	0.012J
Ethylbenzene		0.57J	0.06J		0.012J
Total xylenes		1.0J	0.55J	0.4	0.076J
2-Hexanone			0.05B		
SEMI-VOLATILES					
Phenanthrene	1.6	0.60J			
Anthracene	0.30J				
Di-n-butyl phthalate	0.63B	1.9B			3.6B
Fluoranthene	1.6	0.60J			
Pyrene	1.6	0.9			
Benzo(a)anthracene	0.88				
Benzo(b&k)fluoranthene	1.2				
Benzo(a)pyrene	0.74				
Benzo(ghi)perylene	0.30J				
Indeno(1,2,3-cd)pyrene	0.30J	1			
Chrysene	1				
1,3-Dichlorobenzene				0.8J	
1,4-Dichlorobenzene		1.0J		1.6	
1,2-Dichlorobenzene		0.80J		0.8J	
1,2,4-Trichlorobenzene				3.2	
PESTICIDES and PCBs					
4,4'-DDE		3.0M	1.34M		
4,4'-DDD		12.9M	5.59M		
Data reported by	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.	ERM, Inc.

A - Data taken from 8 December 1986 report

* - Large volume composite

** - Grab samples to obtain preliminary data

B - this analyte was also found in the method blank and is of questionable qualitative significance

J - estimated value

Blanks indicate not detected

M - This pesticide result was comfirmed by GC/MS.

(1,2,3-cd)pyrene were reported in one of the two samples taken from the western swamp area. The source of the PAH's in the ice-house sample (total PAH concentration of 9.26 mg/kg) may be the coal sediment washed from the anthracite region well to the north of the site. The Soil Conservation Survey (SCS) Soil Survey for Montgomery County states that the Rowland silt loam, which occurs in the floodplain of the Schuylkill River, does contain anthracite coal sediment.

Two substituted benzenes, 1,4-dichlorobenzene (1.8 mg/kg) and 1,2,4-trichlorobenzene (3.2 mg/kg) were detected in the July air stripper outfall sample.

Excluding compounds detected in the method blanks five volatile organic compounds were detected in the air stripper outfall samples. 1,2,3-trichloropropane was found in both air stripper samples (0.033 mg/kg and 6.3 mg/kg). Trichloroethylene (0.04 mg/kg) and tetrachloroethylene (.05 mg/kg) were found in the initial air stripper sample along with total xylenes (0.4 mg/kg) and chlorbenzene (0.09 mg/kg).

Pesticides were found only in soil samples collected in the western swamp area. 4,4'-DDD concentrations were 8.59 mg/kg and 12.9 mg/kg; and, DDE concentrations were 1.34 mg/kg and 3 mg/kg.

Inorganic Constituents

Concentrations of inorganic constituents in soil samples taken from the west swamp (SS067, SS069), air stripper outfall (SS068, SS070), and ice-house (SS066), are presented in Table 4-30. Ranges and mean concentrations of these elements commonly

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reported for soils of the eastern United States are presented in
Table 4-18. With the exception of zinc, copper, selenium, and
lead, inorganic constituent concentrations were well within or
below the commonly reported range. Zinc and lead levels in the
initial sample (SS068) from the air stripper outfall were
substantially higher than average levels. This is most likely
attributable to anthropogenic sources of zinc and lead, however,
these sources may not be related to activities at the Tyson's
Site as high levels of zinc, 20-1200 mg/kg, and lead,
218-10,900 mg/kg, are commonly reported for similar areas of
urban development (Preer et.al., 1980). Copper concentrations
exceeded typical levels reported for soils of the eastern United
States (Table 4-18) in the initial sample (SS067) obtained from
the Western Swamp location; selenium exceeded typical levels in
the September sampling at the Western Swamp. Elevated levels of
these constituents, however, have been reported for similar
organic rich soils (Pendias and Pendias, 1984).

Significant variations in the concentration of a number of these
inorganic constituents including aluminum, zinc, lead, barium,
chromium, copper, iron, manganese, nickel and vanadium were found
to exist among sampling locations and between sampling dates.
These variations are thought to be the result of the
heterogeneity of the soils developing on the Schuylkill River
floodplain. These soils, mapped as the Rowland series, exhibit
wide variations in the organic matter content and the thickness
and composition of the sediment layer existing at the soils
surface (Smith and Soil Survey Staff, 1967). In areas high in
organic matter e.g. western swamp, constituents strongly absorbed
by organic matter such as copper and arsenic would be expected to
accumulate (Pendias and Pendias, 1984). Both of these

constituents were present at higher concentrations in the western swamp samples than in either the ice-house or air stripper samples.

With regard to the sediment layer, this layer is derived primarily from coal sediments washed from the anthracite regions of Pennsylvania, north of the sampling area. This layer is reported to vary in thickness from 1 to 3 feet, subsequently, variations in the amount of sediment present may significantly affect the concentrations of inorganic constituents. For example, coal sediments are typically high in iron, soil samples taken from areas with a thicker sediment cap would be expected to exhibit higher iron concentrations than those obtained from areas with a thinner sediment cap. Additionally, certain inorganic constituents are often closely associated with other constituents, such that high concentrations of one element occur in conjunction with high concentrations of others (Pendias and Pendias, 1984). This relationship is particularly true for nickel and manganese which are closely associated with iron. Note that in areas where iron concentrations are high, e.g. western swamp (SS067), nickel and manganese concentrations are also high.

Leachate Generation

Leachate generated from the composite sediment samples collected to the west of the ice-house (background), western swamp, and air stripper discharge ditch were used in acute and chronic flow-through bioassays using juvenile daphnia (Daphnia magna) and newly born fathead minnows (Pimephales promelus). The bioassays were conducted by the Academy of Natural Sciences of

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Philadelphia (Academy). A complete report on procedures and results of this part of the investigation are given in Appendix R.

In addition to the generated leachates, two additional samples were analyzed. These were the dilution water used in the bioassay and a sample of the reference toxicant water which consisted of dilution water plus the reference toxicant (sodium lauryl sulfate). Sample designations and sample descriptions are presented in Table 4-31. Analytical results for inorganic constituents, organic compounds and tentatively identified compounds for the leachate water samples are presented in Table 4-32.

Trace level concentrations of aluminum, iron, lead, manganese, and zinc were detected in the three leachates. The elevated aluminum and iron in LW003 may be due to the fine soil particles associated with this sample which significantly affected the daphnia bioassay due to turbidity in the leachate.

Unqualified HSL organic compounds detected were limited to DDD in Sample LW006 at 0.4 ug/l and DDT in Samples LW006 and LW007 at 0.1 ug/l and 0.2 ug/l, respectively. Ten tentatively identified compounds were detected in the five samples.

Acute and Chronic Bioassays

The bioassays were conducted at the Academy laboratories following established testing protocols. Leachate was generated following ASTM Method D-3987-85. Prior to use in the bioassay,

TABLE 4-31

LEACHATE WATER LOG

Sample #	Date Collected	Sample Description
LW001	8/7/86	Dilution water
LW-002	8/7/86	Reference Toxic
LW-003	8/7/86	Leachate #1 derived from soil collected 1000' west of ice house.
LW-006	9/30/86	Leachate #2 - taken from from western swamp
LW-007	9/30/86	Leachate #3 - air stripper discharge

TABLE 4-32

TYSON'S SITE
LEACHATE WATER RESULTS
HSL ORGANIC COMPOUNDS
(concentration in mg/l, total concentrations)

	LW-001*	LW-002*	LW-003*	LW-006*	LW-007*
	Dilution Water	Reference Toxicant	Background	Western Swamp	Air Stripper
Volatiles					
Acetone	NV	NV	NV	NV	NV
2-Butanone	6 B	6 B	8 B	4 JB	4 JB
Total Xylenes				2 J	
Semi-Volatiles					
Di-n-butyl phthalate		6 J			
Pesticides					
DDE				0.08 J	
DDD				0.4	
DDT				0.1	0.2

B=This analyte was also found in the method blank
J=Estimated value.
Blank=none detected.
*= Locations found on Plate 4.
Q= Did not pass the quality control criteria.
NV= Not valid.

TABLE 4-32

TYSON'S SITE
LEACHATE WATER RESULTS
TENTATIVELY IDENTIFIED COMPOUNDS
(concentration in ug/l, all values estimated)
(continued)

	LW-001*	LW-002*	LW-003*	LW-006*	LW-007*
	Dilution Water	Reference Toxicant	Background	Western Swamp	Air Stripper
Benzene, 1,2-dimethyl				13	
Benzene, methyl					55 B
(Benzene, (1-methyl undecyl)					12
Z-9 octadecen-1-ol				61 B	84 B
Fatty alcohol	36.9 B	646.5	32.6		
Benzene,1,'sulfonylbis	17.5				
Phthalate ester	10.6	33	46.2		
1,2-benzenedicarbon-liz acid ester	10.7				
Benzene, 1,1'-sulfonylbis			16		
Hexadecanoic acid, butyl ester				11	
Total unknowns	77.5	38.9	81.9	13	40
Total aliphatic hydrocarbons	28.2	1066.1			10
Total chlorinated hydrocarbons				62	49

B= This analyte was also found in the blank.
*= Locations found on Plate 4.
Blank= none detected.

TABLE 4-32

**TYSON'S SITE
LEACHATE WATER RESULTS
INORGANIC CONSTITUENTS
(concentration in mg/l, total concentrations)
(continued)**

	LW-001*	LW-002*	LW-003*	LW-006*	LW-007*
	Dilution Water	Reference Toxicant	Background	Western Swamp	Air Stripper
Aluminum			6.7	0.2	0.7
Cadmium			0.019	0.01	0.005
Copper					0.04
Iron			5.36	0.36	0.35
Lead			0.01		0.005
Manganese			0.03	0.7	0.16
Zinc			0.09	0.04	0.09

Blank=none detected
*=Locations found on Plate 4.

the leachate was separated from the sediment by continuous flow centrifugation. The background sample leachate (LW003) was observed to be turbid after centrifugation.

Daphnia testing consisted of 21-day exposure to assess survival (acute) and reproductive (chronic) effects. The fathead minnows were exposed for a 7-day period to assess survival and growth effects.

The fathead minnows did not exhibit a significant difference in survival or growth in any test concentrations of the three leachates in comparison to the dilution water controls.

Daphnia response was more variable, especially in the background leachate tests where the turbidity in the centrifuged leachate appears to have had an effect. It is believed that long-term exposure (more than 6 to 7 days) to the turbidity of the leachate was a factor in affecting the feeding of Daphnia in the test vessels. In retrospect a higher feeding regime should have been used. It is also possible that a chronic effect, emergent only after long-term exposure and independent of the turbidity, was present in the soil leachates. These alternatives could not be explored within the time constraints of the reporting requirement. The lack of any trend in the effects observed in the background sediment leachate testing supports the possibility of an interaction of turbidity and any potentially toxic material in the leachate.

The air stripper leachate was observed not to be visibly turbid. Test results indicated a 21-day LC50 (Lethal Concentration to 50 percent of the test organisms) of 78 percent leachate.

Reproductive effects were observed in the lowest concentration
tested (60 percent leachate).

The West Swamp leachate was more turbid than the air stripper
leachate, but not as turbid as the background leachate. The
acute toxicity, 21-day LC50 was 69.6 percent of the leachate and
a chronic effect on Daphnia was observed at 60 percent leachate.
The air stripper and West Swamp leahcates had a similar toxicity
to Daphnia. No acute or chronic effects were observed for
fishes.

The recommended screen tests to be performed for establishing
test concentrations are acute tests (48 hours for Daphnia and 96
hours for fathead minnows). No acute toxicity was observed;
therefore, high leachate concentrations were used anticipating no
acute toxicity but possibly some chronic toxicity. In fact,
significant acute and chronic toxicity were not seen until Day
13-14 in the Daphnia tests and none in the fish tests. If a
toxicity exists, acute or chronic, long-term exposure of at least
two weeks is necessary. The recommended screen tests were
unfortunately inappropriate for predicting the observed effects.

Because of the excessive turbidity in the background leachate, it
was difficult to differentiate the effects attributable to the
turbidity from the potential toxicity. Due to this problem and
other associated difficulties encountered with the methods
employed, the additional bioassays discussed below were
conducted.

4.6.4.4 Additional Sediment Toxicity Bioassays

As discussed in Section 3.5.3.5 surface sediment samples were collected from seven locations (Plate 8) in the Floodplain Area for the additional sediment toxicity bioassays. The results of analysis of samples of these sediments are provided in Table 4-33.

In general, the results of these analyses are similar to those for samples from those general areas obtained during other phases of work in the floodplain area. Overall, the highest metal concentrations were detected in the samples from the air stripper discharge area and the DDT area. Site related organic compounds such as 1,2,3-trichloropropane, toluene, xylenes, 1,2,4-trichlorobenzene, and 1,4-dichlorobenzene were reported in samples from the air stripper discharge areas and the west swamp, signal tower ditch and DDT area which all receive surface water discharges from the former lagoon area. 1,2,3-trichloropropane (.017 mg/kg) was reported in the gas tank ditch to the east of the air stripper discharge areas. A similar concentration was reported in the sediment samples from this area described in Section 4.6.4.4. PNAs were detected for the west swamp. Only estimated concentrations of PNAs were reported in the DDT area and the air stripper discharge area.

Elevated concentrations of DDD, DDE, and DDT were reported in the samples from the west swamp and DDT area. These two areas are adjacent to each other and may reflect a common source. As stated previously, these pesticides are not present in the former lagoon area. No pesticides were found in the sediment samples

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TABLE 4-33
TYSON'S SITE
FLOODPLAIN AREA SEDIMENT SAMPLE RESULTS
(SEDIMENT TOXICITY BIOASSAYS)
HSL INORGANIC CONSTITUENTS
(Concentration in mg/kg, dry wt. basis)

CONSTITUENTS	BA-001S 5/11/87 gas tank ditch	BA-002S 5/11/87 air stripper	BA-003S 5/11/87 western swamp	BA-004S 5/11/87 signal tower ditch	BA-005S 5/11/87 western ditch	BA-006S 5/11/87 control	BA-007S 5/11/87 DDT area
Aluminum	5480	17100	18400	6410	11900	5120	14400
Arsenic	2.5	13.7	8.2	4.3	5.2	2.6	11.4
Barium	72.2	432	227	106	160	64.8	135
Beryllium	0.40 B	2.2	1.1	0.80	0.70	0.40 B	1.1
Chromium	9.4	27.3	27.4	13.4	14.7	10.2	21.2
Cobalt	4.0	16.4	19.2	6.0	6.5	2.5	10.6
Copper	20.1	98.4	49.3	28.3	24.5	12.7	109
Iron	8660	36100	20900	15800	15200	7850	20900
Lead	22.7	109	126	95.4	55.5	29.2	103
Manganese	229	409	1120	142	462	133	347
Nickel	5.3	21.9	21.9	10.4	8.2	8.9	13.3
Selenium	0.40 B	3	1.9	0.30 B	0.50 B	0.3 B	1.0 B
Vanadium	12.0	60	43.8	11.9	19.6	11.4	37.1
Zinc	77.9	1070	299	69.2	96.1	81.7	182
Cadmium		0.11	0.60	0.20	0.20	0.60 B	0.60
Thallium		3.3 B		0.60 B	1.0 B		1.6 B

Qualifier Codes:
B: This result is of questionable qualitative significance since this constituent was detected in blanks(s) at similar concentrations.

TABLE 4-33 (continued)
TYSON'S SITE
FLOODPLAIN AREA SEDIMENT SAMPLE RESULTS
(SEDIMENT TOXICITY BIOASSAYS)
HSL ORGANIC COMPOUNDS
(Concentration in mg/kg, dry wt. basis)

COMPOUNDS	BA-001S 8/11/87 gas tank ditch	BA-002S 8/11/87 air stripper	BA-003S 8/11/87 western swamp	BA-004S 8/11/87 signal tower ditch	BA-005S 8/11/87 western ditch	BA-006S 8/11/87 control	BA-007S 8/11/87 DDT area
VOLATILES							
1,2,3-Trichloropropane	0.017	0.065	0.183	0.007			
Methylene chloride	0.004 B	0.016 B		0.027 B	0.007 B	0.004 B	
Acetone	0.024 NV	0.44 NV	0.030 NV	1.000 NV	0.082 NV	0.025 NV	0.270 NV
trans- 1,2-Dichloroethene		0.071		0.019			
2-Butanone		0.110		0.010 J	0.010 J		0.053
Trichloroethene				0.009			
4-Methyl-2-pentanone		0.055					
Tetrachloroethene	0.004 J		0.011 J	0.004 J			
Toluene		0.550			0.003 B		0.110
Chlorobenzene		0.250					0.053
Ethylbenzene		0.490					0.110
Total xylenes		7.100		0.021			1.000
Vinyl chloride		0.016 J					
Benzene		0.011 B					
Chloroethane						0.004 J	
SEMI- VOLATILES							
1,2,4-Trichlorobenzene		3.10					
1,2-Dichlorobenzene		2.00 J					0.80 J
1,4-Dichlorobenzene		3.60					
4-Methylphenol		14.00			1.30		
Benzo (a) anthracene				0.30 J	1.35	0.38 J	
Benzo (a) pyrene	0.26 J				1.14	0.38 J	
Benzo (b) fluoranthene	0.26 J			0.30 J	2.17	0.80	
Chrysene	0.40 J			0.45 J	1.47	0.51	
Fluoranthene	0.67			0.65	3.26	1.11	0.53 J
Phenanthrene	0.71			0.64	2.26	0.69	0.53 J
Pyrene	0.53			0.64	2.45	0.60	
1,3-Dichlorobenzene		1.09 J					
Benzoic acid		1.63 J					
Naphthalene				0.30 J	0.33 J		
2-Methyl naphthalene				0.30 J	0.49 J		
Benzo(k)fluoranthene				0.30 J			
2,4-Dimethylphenol					0.33 J		
Dibezofuran					0.33 J		
Fluorene					0.33 J		
Anthracene					0.49 J		
Indeno(1,2,3-cd)pyrene					0.49 J		
Benzo(ghi)perylene					0.49 J		
Di-n-butyl phthalate							0.53 B

Qualifier Codes:
J: This result should be considered a quantitative estimate.
B: This result is of questionable qualitative significance since this constituent was detected in blanks(s) at similar concentrations.
NV: The results for acetone are not valid it was used for a decontamination solvent.

TABLE 4-33 (continued)
TYSON'S SITE
FLOODPLAIN AREA SEDIMENT SAMPLE RESULTS
(SEDIMENT TOXICITY BIOASSAYS)
HSL PESTICIDES
(Concentration in mg/kg, dry wt. basis)

	BA-001S 5/11/87 gas tank ditch	BA-002S 5/11/87 air stripper	BA-003S 5/11/87 western swamp	BA-004S 5/11/87 signal tower ditch	BA-005S 5/11/87 western ditch	BA-006S 5/11/87 control	BA-007S 5/11/87 DDT area
COMPOUNDS							
DDD		0.279	3.26				2.12
DDE			1.56				1.11
DDT			0.123				0.42
PCB-1254				0.150 J	0.300	0.013 J	
PCB-1260					0.64		

Qualifier Codes:

J: This result should be considered a quantitative estimate.

TABLE 4-93 (continued)
TYPO'S SITE
FLOODPLAIN AREA SEDIMENT SAMPLE RESULTS
(SEDIMENT TOXICITY BIOASSAYS)
TENTATIVELY IDENTIFIED COMPOUNDS
(concentration in mg/kg)

	BA-001B 5/11/87	BA-002B 5/11/87	BA-003B 5/11/87	BA-004B 5/11/87	BA-005B 5/11/87	BA-006B 5/11/87	BA-007B 5/11/87
SEMIVOLATILE FRACTION							

Qualifier Codes:
J: This result should be considered a estimated a quantitative estimate.

TABLE 4-33 (continued)
TYSON'S SITE
FLOODPLAIN AREA SEDIMENT SAMPLE RESULTS
(SEDIMENT TOXICITY BIOASSAYS)
GENERAL PARAMETERS
(concentration in mg/kg)

ITEM	UNIT	BA-001S 5/11/87 gas tank ditch	BA-002S 5/11/87 air stripper	BA-003S 5/11/87 western swamp	BA-004S 5/11/87 signal tower ditch	BA-005S 5/11/87 western ditch	BA-006S 5/11/87 control	BA-007S 5/11/87 DDT area
Moisture	% by wt.	25.2	51.7	63.5	32.9	38.7	21.3	52.3
TOC	mg/kg as received	3900	11000	9700	8400	14000	2300	11000
TOC	mg/kg dry wt. basis	4400	60000	27000	9500	23000	2900	29000
pH	1:1	7.75	6.77	7.03	7.18	6.95	6.97	6.82
Particle Size-mesh 4	% passing	85.79	99.97	98.69	99.46	99.22	99.93	99.03
Particle Size-mesh 8	% passing	75.13	99.62	96.36	97.80	98.76	97.47	98.55
Particle Size-mesh 50	% passing	33.25	98.84	93.50	65.78	81.19	27.87	90.07
Particle Size-mesh 200	% passing	26.70	90.15	83.62	48.65	64.63	22.49	79.52

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taken from the discharges to the river described in Section
4.6.4.4. DDD was also detected (0.279 mg/kg) in the air stripper
discharge area. PCB-1254 and PCB-1260 were detected at 0.30
mg/kg and 0.64 mg/kg, respectively, in the western ditch which
does not receive runoff from the former lagoon area. No PCBs
were reported in a sediment sample taken from this areas as part
of the discharge to the river study; Section 4.6.4.4. An
estimated concentration of PCB-1254 (.015 mg/kg) was reported in
the signal tower ditch sample.

Table 4-33 also includes the results of the % moisture, TOC, and
grain size analysis of the sediment samples. As noted, those
parameters vary greatly for the seven sediment samples.

Results of the 48-hour liquid phase elutriate test indicated no
mortality in any of the 21 test chambers or triplicate dilution
water controls. The results suggest that the site sediment
elutriates tested do not have an acutely toxic potential to
Daphnia.

Results of the 48-hour survivorship tests using 5-day old Daphnia
in the solid phase sediment and water beaker test indicate one
(1) death in the control sediment (Sample 6) triplicate sample.

The ten (10) day adult survivorship test using the solid phase
sediment and water beaker test showed that surviving adults did
not display a significant difference (P>0.918) among the
chambers containing the test sediment samples, control sediment
sample, and dilution water control. Total number of animals
counted were:

Station	Total Daphnia	Range
1 Gas Tank Ditch	1107	303-419
2 Air Stripper	1916	583-718
3 West Swamp	950	221-382
4 Signal Tower Ditch	1422	442-490
5 West Nest 4 Ditch	1945	597-722
6 Control Sediment	1914	566-714
7 DDT Suspect Area	1228	307-474
8 Dilution Water - Schuylkill River	1618	452-601

Significant differences were observed, however, among the total number of animals (surviving adults plus progeny) at the end of 10 days of exposure compared to the control soil samples. Samples 1 (1107 Daphnia), 3 (950 Daphnia), and 7 (1228 Daphnia) had significantly fewer animals than the control soil (1914 Daphnia).

Comparing samples 1, 3, and 7 to the river water control (1618 Daphnia) indicated that samples 1 and 3 were significantly different.

Differences in the control sediment (1914 Daphnia) and the river water control (1618 Daphnia) may be due to nutrient (phosphorous and nitrate) stimulation of the algae during the test.

A summary of the quality assured analytical data of both studies is presented in Tables 4-34 and 4-35. Also included are USEPA ambient water quality criteria, and several lowest observed effects levels.

Results of the liquid phase elutriate chemical analysis indicate no potential acute toxicity based on chemical analysis. The LOEL value for 1,2,3-trichloropropane is at least 1000 times more than the highest concentration detected of 1,2,3-trichloropropane. Two metabolites of DDT, DDE and DDD were detected in the swamp sample (3) at concentrations of less than part per billion.

Analytic results of the solid phase sediment and water beaker test were more variable. Sediments 2, 4, 5, 6, and 7 leachate exceed the chronic criteria for iron. Only one of the iron analytical data is not a quantitative estimate. Sediment 2 had an estimated 16 mg/l iron, however sediment 2 leachate supported the same number of Daphnia as the control sediment.

Iron concentration differences in the two types of leachate may be due to conditions established in the test chambers during the three-day settling period in the water beaker test.

The PCB-1248 concentration in Sediment 3 leachate exceeded both the chronic and acute criteria. Sediment 3 leachate also had the lowest total number of Daphnia at the end of the test period. No

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TABLE 4-34

ANALYTIC RESULTS - SEDIMENT BIOASSAY

LIQUID PHASE ELUTRIATE METHOD

mg/l

Station No. Description	1 Gas Test Ditch	2 Air Stripper	3 West Swamp	4 Signal Tower Ditch	5 Western Ditch	6 Control	7 DDT Area	8 Schuylkill River	Water Quality Criteria For Protecting Aquatic Life (a) Acute	Chronic
Aluminum										
Barium	0.2	0.2		0.1	0.3	0.2	0.2	0.1		
Iron	1.33J	0.87J	0.43J	0.33J	0.57J	0.1	0.2	0.25J		1.0
Magnese	0.048	0.65	0.02	0.68	3.75	0.18J	4.9J	0.05		
Zinc	0.012	0.028	0.028	0.018	0.028	1.55	1.38	0.018	0.32	0.047
1,2,3-Tri-chloropropane		0.073	0.028	0.029		0.028	0.036		42.0 LOEL b,c	
Chloroform	0.0038		0.0038	1.002B		0.0028			28.9 LOEL	1.24 LOEL
Total Xylenes		0.011				0.004J				
1,4-Dichloro-benzene		0.006J							1.12 LOEL	0.763 LOEL
1,2-Dichloro-benzene		0.005J							1.12 LOEL	0.763 LOEL
Butylbenzyl phthalate			0.0035						0.94 LOEL	0.003 LOEL
DDE			0.00005J						1.05 LOEL	
DDD			0.0001						0.0011	0.000001

B - This result is of questionable qualitative significance since this constituent was also detected in blank(s).

J - This result should be considered a quantitative estimate.

a. Source: USEPA 1986 Quality Criteria for Water
b. Lowest Observed Effects Level
c. Source: USEPA 1985 Chemical and Physical Properties of Compounds Present at Hazardous Waste Sites

TABLE 4-35

ANALYTIC RESULTS - SEDIMENT BIOASSAY

SOLID PHASE SEDIMENT AND WATER BEAKER TEST

mg/l

Station No. Description	1 Gas Tank Ditch	2 Air Stripper	3 West Sump	4 Signal Tower Ditch	5 Western Ditch	6 Western Control	7 DDT Area	8 Schuylkill River	Water Quality Criteria For Protecting Aquatic Life (a) Acute	Chronic
Aluminum	0.1	0.1								
Arsenic		0.011							0.36	0.19
Barium	0.2	0.4	0.2		0.2	6.8				
Chromium				0.2	0.2	0.1	0.01		0.16	0.011 / 1.0
Iron	0.63	16.4J	0.42J	3.15J	1.65J	1.5J	1.6J			
Manganese	0.54	0.67	0.18	0.76	2.04	2.79	0.64			
Zinc	0.048	0.20B	0.028	0.028	0.02	0.006			0.32	0.047
1,2,3-Trichloro-propane	0.005	0.004J	0.018	0.006	0.006				42.0 LOEL b,c	
Toluene		0.009							17.5 LOEL	
4-Methyl-2-Pentanone		0.002J								
Chlorobenzene		0.002J							0.25 LOEL	LOEL
Ethylbenzene		0.002J							32.0 LOEL	
Total Xylenes		0.052								
Phenol		0.006J							10.2 LOEL	2.56 LOEL
1,4-Dichloro-benzene									1.12 LOEL	0.763 LOEL
1,2-Dichloro-benzene		0.007J							1.12 LOEL	0.763 LOEL
4-Methylphenol		0.100								
Bis(2-ethyl hexyl)phthalate		0.0068					0.0002		0.94 LOEL	0.003 LOEL
DDD			0.0002						0.0011	0.000001
PCB 1248			0.0035						0.002	0.000004

B - This result is of questionable qualitative significance since this constituent was also detected in blank(s).

J - This result should be considered a quantitative estimate.

a. Source: USEPA 1986 Quality Criteria for Water

b. Lowest Observed Effects Level

c. Source: USEPA 1985 Chemical and Physical Properties of Compounds Present at Hazardous Waste Sites

PCBs were detected in the liquid phase elutriate of sediment 3 or any other sediment in both test types. Sediment 3 leachate also had sub-part per billion DDD levels as did sediment 1. PCBs are not site related compounds as they have not been reported in samples taken from the former lagoons.

Based on chemical analysis, 10 day effects on survival and reproduction on Daphnia in Sediment 3 (west swamp), and Sediment 7 (DDT area) may be related to the metabolites of DDT. Sediment 1 (gas tank ditch) supported to second lowest total number of Daphnia. None of the chemical parameters measured on Sediment 1 can be suggested as the toxic agent. As with the PCBs, the pesticides are not considered site related compounds as they have not been reported in samples taken from the former lagoons. A complete report on these additional bioassays is provided in Appendix S.

4.7 Comparison of Organic Compounds Detected in On-Site and Off-Site Samples

An extensive data base exists for the organic compounds and inorganic constituents in the former lagoon area. This includes analysis of subsurface and surface soil samples obtained during the On-Site RI, (Appendix A), the Woodward Clyde Consultants Supplemental Soil Investigation (Appendix B), and the SRW investigation of the area west of the former lagoon area (Appendix C). Surface and subsurface soil and sediment and surface water samples from several of the Off-Site areas were also collected during the On-Site RI.

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Table 4-36 is a comparison of the organic compounds detected in
the former lagoon areas during the above investigations and the
organic compounds detected in the Off-Site Operable Units during
the On-Site RI and the Off-Site Operable Unit RI. A broad suite
of similar organic compounds were detected in both the former
lagoon areas and the various Off-Site Operable Units. However,
it is also quite obvious from Table 4-20 that the PAHs detected
during the various investigations did not originate from the
former lagoons.

Possible sources of the PAHs to the Off-Site Operable Units
include the following:

- coal fines washed downriver from coal crushing/washing
  and storage operations along the northern reaches of
  the river;

- burning of construction materials;

- bottom ash used as fill material for the railroad
  ballast;

- materials used for maintenance and construction of the
  railroad;

- spills of coal, coal related products, and chemicals
  during the transport of these materials via the
  railroad;

- fly ash and gaseous emissions from the coal fired
  generating station on Barbadoes Island.

COMPOUND	ON-SITE ORGANIC COMPOUNDS	EPA OFF-SITE INVESTIGATION	EPA OFF-SITE INVESTIGATION OPERABLE UNITS					DEEP
			HILLSIDE	RAILROAD FLOODPLAIN WATER** SOILS	SEDIMENT		DEEP AQUIFER
2-CHLOROPHENOL	X						
2,4-DIMETHYLPHENOL	X		X				
PHENOL	X	X	X				X
2-METHYLPHENOL	X		X		X	X	
4-METHYLPHENOL	X	X	X		X X	X	
BENZENE	X	X		X X	X	X	X
CHLOROBENZENE	X	X	X	X X	X		X
ETHYLBENZENE	X	X	X	X X	X		X
1,2,4-TRICHLOROBENZENE	X	X		X X	X		X
1,2-DICHLOROBENZENE	X	X		X	X		X
1,3-DICHLOROBENZENE	X			X	X		
1,4-DICHLOROBENZENE	X	X		X X	X		
NITROBENZENE	X			X			X
NAPHTHALENE	X	X	X	X X	X	X	
2-METHYLNAPHTHALENE	X	X	X	X X	X	X	
2-CHLORONAPHTHALENE	X		X				
N-NITROSODIPHENYLAMINE	X	X		X			
BIS(2-ETHYLHEXYL)PHTHALATE	X	X		X	X		
DI-N-BUTYL PHTHALATE	X	X					
DI-N-OCTYL PHTHALATE	X	X			X		
DIETHYL PHTHALATE	X	X					
BUTYL BENZYL PHTHALATE	X						
ANILINE	X						
METHYLENE CHLORIDE	X	X					
FLUOROTRICHLOROMETHANE	X						
TETRACHLOROETHENE	X	X	X	X X	X	X	X
TOLUENE	X	X		X	X	X	X
TRICHLOROETHENE	X	X	X	X X	X	X	X
TOTAL XYLENE	X	X		X X	X	X	X
1,1-DICHLOROETHANE	X	X		X X	X		X
CHLOROFORM	X	X		X	X		X
TRANS-1,2-DICHLOROETHENE	X	X		X X	X		X
TRANS-1,3-DICHLOROPROPENE	X	X					
ACETONE	X	X					X
4-METHYL-2-PENTANONE	X	X			X	X	X
HEPTACHLOR EPOXIDE	X						
1,2,3-TRICHLOROPROPANE	X	X	X	X X	X	X	X
2-BUTANONE	X				X		
1,1,2,2-TETRACHLOROETHANE	X			X			
VINYL CHLORIDE	X						
ALDRIN #	X			X			X
4,4'-DDE #	X	X		X			
4,4'-DDD #	X	X		X		X	
4,4'-DDT #	X	X	X			X	
ENDOSULFAN I #	X		X				X
ENDOSULFAN II #	X			X			X
ENDOSULFAN SULFATE #	X		X				X
HEPTACHLOR #	X						
ALPHA-BHC #	X						
GAMMA-BHC-LINDANE #	X						X
BETA-BHC #	X	X			X		X
PCB (1254,1260) #	X	X					
DIELDRIN #	X	X					X
DELTA-BHC #	X						

THE FOLLOWING COMPOUNDS WERE DETECTED IN THE OFF-SITE OPERABLE UNITS ONLY AND THUS ARE NOT RELATED TO THE TYSON'S SUPERFUND SITE.

CARBON DISULFIDE *	TD (NOT DETECTED)							
2-HEXANONE *	TD (NOT DETECTED)							
1,1,1-TRICHLOROETHANE				X				X
CIS-1,3-DICHLOROPROPENE								X
1,2-DICHLOROPROPANE		X						X
DIBENZOFURAN *		X(TD)	X	X	X			X
BENZOIC ACID		X(TD)						X
N-NITROSODI-N-PROPYLAMINE		X						
CHLORDANE		X						
FLUORANTHENE		X	X	X	X	X	X	X
ISOPHORONE		X						
ACENAPHTHENE		X			X	X		
ACENAPHTHYLENE					X	X		
PYRENE		X	X	X	X	X	X	X
PHENANTHRENE		X	X	X	X	X	X	X
BENZO(A)ANTHRACENE		X	X	X	X	X	X	X
BENZO(B)FLUORANTHENE		X	X##	X##	X##	X##	X##	X##
CHRYSENE		X	X	X	X	X	X	X
ANTHRACENE		X		X	X	X	X	
BENZO(A)PYRENE		X		X	X	X	X	
BENZO(K)FLUORANTHENE		X				X		
BENZO(G,H,I)PERYLENE		X			X	X	X	X
FLUORENE		X			X	X	X	
DIBENZO(A,H)ANTHRACENE		X				X	X	
INDENO(1,2,3-CD)PYRENE		X			X	X	X	X

* HSL COMPOUND IN EPA INVESTIGATION BUT TENATIVELY IDENTIFIED COMPOUND IN EPA INVESTIGATION
TD - TENATIVELY IDENTIFIED COMPOUND
** Includes surface water samples from the schuylkill river and ground water
samples taken from the EPA wells completed in the unconsolidated deposits on the floodplain.
Detected in background equivalent samples only or at higher concentrations upgradient
than on-site. These compounds are not related to the Tyson's Superfund Site.
Indicates both benzo(b)fluoranthene and benzo(k)fluoranthene

AR301604

APPENDIX G

DETERMINATION OF CARCINOGENIC
POTENCY FACTORS FOR
1,2,3-TRICHLOROPROPANE

Attachment A

SUBJECT: Current status of 1,2,3-TCP in telephone
conversation with Dr. Tom Burko

DATE: APR 0 7 1987

FROM: Richard L. Brunker, Toxicologist
Site Support Section (3HW26)

TO: Tim Travers, Environmental Scientist
Sara Special Site Section (3HW17)

In a telephone conversation with Dr. Tom Burko of the National
Toxicology Program, I was made aware of the issuance of an "Alert" letter
concerning the analysis of the pathological results of animal studies of
the effects of 1,2,3-trichloropropane. I was told that the laboratory
studies in question yielded data indicating significantly higher numbers
of tumors in experimental animals. The incidence of tumors was found to
be dose dependent.

The examination of the animals from rat studies revealed increased
numbers of tumors on the tongue of experimental animals. Tested mice
developed significantly higher numbers of stomach tumors.

(The fact that two different species are affected and the numbers of
tumors increased with higher doses is usually the criteria for designating
a substance a B2 carcinogen. This category is equivalent to designating
a substance as a probable human carcinogen. It is important in contacts
with responsible parties to remember that no final decision in this regard
has yet been announced.)

The preceeding revelations are in concord with, and support, our
current position that there is compelling information and data that we
should consider this substance as a carcinogenic threat to humans.

Dr. Burko also said that number of tumors are such that pathologists'
examinations of tumors for type (benign or malignant) will not affect the
eventual findings. He feels that there is no question concerning the
carcinogenicity of 1,2,3-TCP in laboratory animals and that the EPA will
agree with this assessment.

AR301606

Date: _May 6, 1987_

Time: _3:15 pm - ___

Name: _Dr. Richard Brunker_

Subject: _Potency factor for TCP (Typens)_

W.O. NO.: _____

Company: _EPA_

Telephone: _____

☞ **Notes** _Dick Brunker called me to say that while the position paper on estimating potency factors for 1,1,1-trichloropropane was well researched and written, he and Dr. Bruce Mulbolt (FDA toxicologists) do not agree. They felt that including class C carcinogens in the data set used to calculate potency factors (q) was inappropriate. They also disagreed with the omission of brominated alkenes. Dick pointed out that bromine compounds are not always more carcinogenic — he noted for example that bromoform and dibromomethane are not carcinogenic while the chloro analogs are. I pointed out that for those compounds that are carcinogenic, the bromo derivatives are much more potent as documented by John Weisburger and Gary Williams._

Dick expressed concern that NTP's Tom Burke would not agree with my estimate and that FDA would appear to be less than diligent when (not if) the CAG comes out with a much higher value. He felt the estimate would appear as a purposeful attempt to underestimate. I expressed some concern about the NTP's ability to assume potency on the basis of non-historical yield data (unreviewed tumor number comparisons).

Dick and Bruce Mulbolt felt that DBCP was essential for inclusion and that they would insist. Dick calculated an oral value of 2.3 × 10⁻¹ but agreed that a method of using geometric mean rather than arithmetic mean would suffice. I suggested that if FDA q controversial we not include it. When I asked about inhalation potency, Dick agreed that the position used would suffice but he would consider alternatives if I desired.

☞ **Action Items**

	Who	Date

Message Received By: _K E Symons_ ☞

Follow-Up: YES _____ NO _____

By Whom: _____

Date: _____

Copies To: File Copy _____

2 _____

3 _____

4 _____

Notes: _____

Environmental Resources Management, Inc.

999 West Chester Pike • West Chester, Pennsylvania 19382 • (215) 696-9110 • Telex 4900007538

12 May 1987

Dr. Richard Brunker, Toxicologist
Site Investigation & Support Section
US EPA, Region III
841 Chestnut Building
Philadelphia, PA 19107   File No.: 272-11

Dear Dr. Brunker:

As discussed in the 10 April 1987 meeting with CIBA-GEIGY Corp
and EPA, Environmental Resources Management, Inc., (ERM) has
prepared this position document on the carcinogenic status of
1,2,3-trichloropropane (TCP). This report is being submitted by
ERM on behalf of CIBA-GEIGY Corp. The following sections of this
document include background information on the compound,
considerations for estimation of an oral carcinogenic potency
factor for TCP, derivation of an oral potency factor for TCP, and
calculation of the inhalation potency factor.

An oral potency factor of 1.0×10^{-1} (mg/kg/day)$^{-1}$ has been
derived for TCP for the purposes of risk quantitations. An
inhalation potency factor equivalent to that approximated for
oral exposure is suggested on the basis of the available data
concerning related compounds.

BACKGROUND

1,2,3-Trichloropropane (TCP) is not included in EPA's
categorization of chemicals evaluated for carcinogenic evidence,
due to an insufficiency of appropriate data. The National
Toxicology Program (NTP), however, is currently conducting a
bioassay of TCP (gavage) in both sexes of the B6C3F1 mouse and
Fischer 344 rat. The 130-week dosing phase of the chronic study
is expected to reach completion in June of 1987. Because the
pathology evaluation stage of the bioassay is on an accelerated
schedule, gross necropsy, histologic examination, statistical
analysis, and data interpretation may require only an additional
6 months instead of the usual one year. Sometime thereafter, a
preliminary draft of the findings will be made available to the
National Toxicology Program Technical Review Subcommittee and to
external reviewers for rigorous evaluation. Assuming no serious
shortcomings in the adequacy of the study protocols, a final
determination of the carcinogenicity of TCP may be anticipated in
1988.

An NPL site with TCP as a major environmental contaminant must
address the issue of carcinogenicity as a critical part of
rigidly scheduled RI/FS processes before the results of the NTP

bioassay can be properly evaluated, finalized, and released. The NTP, however, has at this time obtained some preliminary data (from morbid and planned 65-week sacrifice animals) which suggest dose-dependent tumorigenic activity of TCP in various organs of both mice and rats. Details concerning the mid-course study results are unavailable for any independent assessment prior to completion and internal audit. However, the NTP regards the preliminary findings as sufficiently compelling to issue an "Early Alert", which was expected to be released before May, 1987.

On the basis of preliminary conclusions by NTP regarding the 65-week rodent data for TCP, regional EPA has adopted the position that TCP should be regarded as a probable human carcinogen (see Attachment A). It is important to emphasize the following points in this context:

1) EPA toxicologists have conservatively assumed a Group B2 classification on the basis of a reasonable expectation of potential adverse human health effects (i.e., NTP informed them of a dose-related increase in tumor incidence in more than one species and in diverse organs/tissues).

2) Virtually no specific information could be provided by NTP at this time to enable a critical review of the bioassay design, conduct, or interpretation of the mid-course findings.

3) The conclusions based on the NTP mid-course data are preliminary and could change as a consequence of evaluating all data at the conclusion of the study or as a result of a subsequent audit.

The requirements of the on-going RI/FS processes include assessing risks associated with potential exposures to TCP. Risk assessments consider not only the qualitative classification of TCP as a carcinogen, but must also generate quantitative estimates of lifetime cancer risk. For potential carcinogens risks are estimated as probabilities. A potency factor, which is an upper 95 percent confidence limit on the probability of a response per unit intake of a chemical over a lifetime, relates estimated exposures to predicted incremental risk. Potency

factors or unit cancer risk values are generally calculated from
dose-response data obtained from animal bioassays and
extrapolated to low-level human equivalent exposure-risk
predictions. However, the laboratory-generated data needed to
derive a carcinogenic potency factor for TCP are unavailable from
the NTP. In order to meet the deadlines of submitting the
TCP-related feasibility studies and conforming to the guidelines
set forth in the Superfund Public Health Evaluation Manual, it is
necessary to generate a carcinogenic potency factor for TCP on
the basis of the best existing data.[1]

CONSIDERATIONS FOR ESTIMATION OF AN ORAL CARCINOGENIC POTENCY FACTOR FOR 1,2,3-TRICHLOROPROPANE

TCP is a member of the halogenated aliphatic class of compounds
which include some of the most widely used and environmentally
encountered chemical substances. In part because of this,
bioassay programs have focused a significant portion of their
efforts on chemicals closely related to TCP. A reasonably
adequate data base on a series of halogenated hydrocarbons tested
in a relatively uniform way has been developed in recent years.
In spite of this, it is not yet possible to accurately predict
the carcinogenic activity of a particular halogenated hydrocarbon
on the basis of data obtained with others.

Ordinarily, not all chemicals that belong to any class are
carcinogenic, nor are all those compounds within a class which
exhibit carcinogenicity equally potent.[2] With the exception of a
few direct-acting agents, the organic carcinogens require
metabolic activation in order to exert their cancer-inducing
properties.[2,4,5] Metabolic activation of procarcinogens to
ultimate carcinogenic forms is generally thought to involve the
production of electrophilic reactants that covalently bind with
target intracellular macromolecules such as DNA and proteins.[4]
In general, structurally related compounds which are metabolized
via similar pathways, are most likely to undergo similar
bioactivation reactions even in relatively very small yield to
produce comparable electrophilic (genotoxic) intermediates.

In comparing TCP with other carcinogenic halogenated
hydrocarbons, there would appear to exist a sufficient body of
information for a series of related chlorinated alkanes to
provide a reasonable data base from which to estimate a

carcinogenic potency factor for TCP. While compounds such as trichloroethylene (TCE) exhibit some similarities with TCP, the chlorinated olefins may be metabolically quite distinct from alkanes. The presence of an unsaturated bond between the carbon atoms of vinyl chloride, TCE, etc., can be expected to introduce a major influence with respect to patterns of metabolic disposition (e.g., reactive epoxide or oxirane intermediates).[6,7] The stability (i.e., reactivity vs. half-life) of the ultimate carcinogenic species is likely to be influenced by the unsaturated carbon bond as well.

In considering only simple halogenated alkanes for correlation with TCP, it is apparent that substantial differences in chemical properties exist between fluoro, chloro, bromo, and iodo alkanes. Chemically, organic bromine is a better leaving group in nucleophilic substitution reactions than chlorine.[8] Brominated alkanes are more reactive. Moreover, notable differences among the haloalkanes have been determined regarding inducibility of hepatic mixed-function oxidase enzymes and covalent binding to microsomal protein, microsomal lipid, or calf thymus DNA.[9] With equal chemical structure, bromo derivatives are generally more toxic than the corresponding chloro compounds.[5] This appears to apply to relative carcinogenic potency as well.

For example, two well-studied analogs, 1,2-dichloroethane and 1,2-dibromoethane, exhibit many qualitative similarities,[10] but the bromo analog is much more mutagenic and carcinogenic.[5] The carcinogenic potency factor calculated for 1,2-dibromoethane is nearly 3 orders of magnitude greater than the estimated value for 1,2-dichloroethane.[1] Oral administration of 1,2-dibromoethane induces (in addition to stomach squamous cell carcinoma) a similar tumor pattern exhibited by the chloro compound (viz., lung and liver tumors in mice, angiosarcomas in male rats, and mammary adenocarcinomas in female rats). The expression time is significantly shorter for the bromo derivative, testifying to its greater potency.[5] Inhalation of 1,2-dibromoethane induces cancer in the nasal cavity. Inhalation results for 1,2-dichloroethane in rats and mice so far are negative, and the difference has been postulated to be due to use of a dose less than the MTD. Unlike the dichloroalkanes, however, some dibromoalkanes appear to be able to react directly with important cellular receptors since they are mutagenic in certain in vitro assay systems without the addition of a liver S-9 fraction.[10]

Brominated hydrocarbons may yet prove to be good qualitative
indicators of carcinogenic activity for corresponding chlorinated
hydrocarbons. Quantitatively, however, the bromo and chloro
alkanes are not equivalent.

Accordingly, among the numerous halogenated aliphatic compounds
which have been tested and thoroughly evaluated for carcinogenic
activity, the simple (unsubstituted) chloroalkanes are most
likely to provide the best representation for deriving a potency
factor for the chloroalkane, TCP. A sufficient number of
carcinogenic compounds in this subclass have been characterized
to constitute a reasonably good and logical data set.

However, since the early NTP data suggest that TCP may exhibit a
greater potency then other chloroalkanes, it is considered
appropriate to include the brominated chloropropane (DBCP) for
purposes of comparison. This is consistent with a conservative
approach to assessing health hazards.

DERIVATION OF AN ORAL POTENCY FACTOR FOR TCP

Only chloroalkanes and the bromochloropropanes which have been
classified as (Group B) and which have published potency factors
(q*) were selected for inclusion in the data set. These related
carcinogens and the most recent potency factors developed by
EPA's Carcinogen Assessment Group (CAG) are listed in Table 1.[1]

Any of several mathematical treatments of the listed (q*) values
can be applied to derive a representative value for this subclass
of carcinogens. However, since tumor induction is generally
considered to be a multi-stage process, (each stage under
independent genetic control),[5] a log-normal distribution of
potencies among the series of compounds is assumed. In such a
multi-variant circumstance a geometric mean is generally believed
to provide the most representative value. The median is also
presented in Table 1. The close agreement between the mean
and the median supports the derivation as a fair representation
of the q* values provided by this set of compounds.

DERIVATION OF AN INHALATION POTENCY FACTOR FOR TCP

Only 2 chloroalkane compounds have been evaluated for inhalation
potency (values published in the Superfund Public Health

Evaluation Manual). In view of the very meager data base it is difficult to estimate a unit cancer risk for the inhalation exposure to TCP with any measure of confidence.

One technique that has been utilized by EPA's Carcinogen Assessment Group is to indirectly estimate inhalation potency factors based on a comparison of the exposure route potency ratios of closely related compounds that have both oral and inhalation data available.[11] The published oral and inhalation carcinogenic potency factors for the related chloroalkanes, dichloromethane and 1,2-dichloroethane, are listed in Table 2.[1] Inhalation potency factors can be calculated for TCP by using the ratios for the two aforementioned substances, according to the following equation:

$$P(TCP,I) = \frac{P(Dichloroalkane, I)}{P(Dichloroalkane, O)} \times P(TCP, O_{est}),$$

The inhalation potency for TCP is estimated to be 1.9×10^{-1} (mg/kg/day)$^{-1}$ using the dichloromethane data, and 3.9×10^{-2} (mg/kg/day)$^{-1}$ using the 1,2-dichloroethane data. Thus, in one case the inhalation potency is estimated to be higher than the oral potency, and using data from another related analog it is lower. On the average, the potencies would be roughly equivalent. Considering the manifold uncertainties inherent in this highly conjectural, but necessary, exercise, it is suggested that the inhalation potency for TCP is conservatively the same as its derived oral potency (i.e., 1.0×10^{-1} per mg/kg/day, risk-exposure units).

Please call me at (215) 692-8606 if you have any questions or require any additional information.

Sincerely,

Rudolf M. Schulh fr

Ken Symms

KS:aek
cc: Karline Tierney, CIBA-GEIGY Corp.

TABLE 1

Carcinogenic Alkane	Oral Carcinogenic Potency Factor (q^*) $(mg/kg/day)^{-1}$
Dichloromethane	7.5×10^{-3}
Chloroform	8.1×10^{-2}
1,2-Dichloroethane	9.1×10^{-2}
Carbon Tetrachloride	1.3×10^{-1}
1,2-Dibromo-3-chloropropane*	1.4×10^{0}
Median	0.91×10^{-1}
Geometric Mean	1.0×10^{-1}

*The Oral potency factor for DBCP has been published in the Federal Register, Vol. 50, No. 219, Wed., Nov. 13, 1985, p. 46982; and in the EPA's most recently revised Health Advisories for 53 chemicals.

Table 2.

Carcinogenic chloroalkane	Oral Potency Factor $(mg/kg/day)^{-1}$	Inhalation Potency Factor $(mg/kg/day)^{-1}$	Ratio $\dfrac{q*\ \text{Inhalation}}{q*\ \text{Oral}}$
Dichloromethane	7.5×10^{-3}	1.43×10^{-2}	1.91
1,2-Dichloroethane	9.1×10^{-2}	3.5×10^{-2}	0.39

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AR301617

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
REGION III
841 Chestnut Building
Philadelphia, Pennsylvania 19107

SUBJECT: The Status of the Interim Potency
Index for 1,2,3-Trichloropropane   **DATE:** JUL 11 87

FROM: Richard L. Brunker, Toxicologist,
Site Support Section (3HW26)

TO: Tim Travers, Acting Chief
SARA Special Site Section (3HW17)

The purpose of this communication is to define the current status of the interim potency index for 1,2,3-trichloropropane. This potency index (1.8×10^{-1} mg/kg/day) was calculated by Dr. Kenneth Symms of ERM Inc. after consultations with Dr. Bruce Molholt of our Enforcement Branch and myself.

This index was calculated, at my suggestion, to provide the PRP with an interim tool in order to carry out the calculations necessary in the formulation of necessary risk assessments in the RI/FS phase of remedial studies. Without such a number progress regarding risk assessment calculations would not include the calculation of risk from the most dangerous and important contaminant (1,2,3-TCP) and progress would have been affected.

It is important to remember that 1,2,3-TCP has been demonstrated to cause considerable numbers of tumors in laboratory animals and appears to fulfill the necessary criteria for a probable human carcinogen (B_2) characterization. The potency index provided represents the arithmetic mean of similar animal carcinogens which is a reasonable strategy in such calculations.

It is also probable that the official potency index that is eventually provided by the Cancer Assessment Group (CAG) for 1,2,3-TCP will differ somewhat from the interim number and a simple factor will be needed to correct for this difference in past calculations in this regard.

Please remember that the calculation of this interim number and its use was allowed in a cooperative spirit to provide the PRP a venue for the calculation of carcinogenic risk from 1,2,3-TCP and enable the risk assessment process for this facility to move forward.

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JUL 22 1987

SARA, Special Site

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APPENDIX H

TOXICOLOGY PROFILES
FOR THE
INDICATOR CHEMICALS
THE TYSON'S SITE OFF-SITE RI
MONTGOMERY COUNTY, PA

APPENDIX H

H.1 Arsenic (USEPA, 1984)

H.1.1 Summary of Health Effects Data

The toxicity of arsenic depends upon its chemical form and the route and duration of exposure. In general, arsenites (As^{+3}) are more toxic than arsenates (As^{+5}), soluble compounds are more toxic than insoluble compounds, and inorganic compounds are more toxic than organic derivatives. Short-term effects of arsenic poisoning are similar in humans and animals. With oral exposure, symptoms include muscular cramps, facial edema, gastrointestinal damage, vomiting, diarrhea, and general vascular collapse. Long-term exposure produces effects similar to those observed following short-term exposure, including damage to the hematopoietic, renal, and nervous systems. In humans, chronic exposure to arsenic is associated with a characteristic pattern of skin lesions. One of the most characteristic effects of chronic arsenic exposure in humans is a pattern of skin disorders, beginning with hyperpigmentation and keratosis, developing in some cases into squamous cell or basal cell carcinoma. Most humans can tolerate the dose of 6.8 mg As^{+3}/day with no adverse effects. The estimated dose of arsenic due to accidental exposure via ingestion of arsenic contaminated foods was about 3.5 mg/day for about 33 days.

H.1.2 Toxic and Carcinogenic Effects

Acute symptoms of arsenic poisoning are similar in both man and experimental animals. The acute symptoms associated with oral exposure to high doses of arsenic include severe gastrointestinal damage resulting in vomiting and diarrhea and general vascular collapse, leading to shock, coma, and death. Other acute effects are muscular cramps, facial edema, and cardiovascular reactions. Airborne exposure at high levels also results in severe irritation of the nasal mucosa, larynx, and bronchi. An aftereffect of acute contact with inorganic arsenic includes peripheral nervous system disturbances and slow recovery and reversible effects on the hematopoitic system. Levels of exposure associated with acute arsenic toxicity vary with the valence form of the element, the trivalent state being approximately 4-fold more toxic than the pentavalent arsenic. Oral LD_{50} values for trivalent arsenic vary from 15 to 293 mg/kg body weight in rats, and from 10-150 mg/kg in other test species.

The collective evidence at the present time for the etiological role of inorganic arsenic in human cancers is strongest for the cancers of the skin and lung. In man, chronic oral exposure to arsenic induces a series of changes in the skin epithelium, proceeding from hyperpigmentation to hyperkeratosis, characterized as keratin and proliferation of the verrucose nature, and leading in some cases to late onset skin cancers.

Although several studies conducted in Taiwan, Argentina, Chile, and Europe have shown an association between elevated levels of arsenic in drinking water and excess skin cancer, such an association has not been observed in several epidemiologic studies conducted in the U.S. EPA is currently reviewing its position on the carcinogenicity of arsenic.

H.1.3 Applicable Standards

The recognized applicable and relevant standards for arsenic are summarized in Table H-1. The ambient water quality criterion for the protection of fresh water life is 0.19 mg/L. A proposed maximum concentration level in drinking water (MCL) has been established at 0.05 mg/L for arsenic. A proposed MCLG (goal) has been established at the same level. Regulations for work place exposures have been developed by OSHA and ACGIH at 0.01 mg/m^3. EPA is currently reviewing its position on the carcinogenicity of ingested arsenic. In establishing an MCLG, EPA has apparently treated arsenic as not carcinogenic by ingestion.

H.2 Barium (USEPA September 1985a, USEPA September 1985b)

H.2.1 Summary of Health Effects Data

Acute barium toxicity is a associated with hypokalemia and electrocardiographic changes as well as other symptoms. The acute threshold toxic dose of barium has been estimated to be 2.9 to 7.1 mg/kg for a 70 kg adult. Several investigations concluded that there was significant negative correlation between (1) barium in drinking water and atherosclerotic heart disease; (2) barium and mortality rates; and (3) and barium and congenital malformations. There appears to be no statistically significant difference in blood pressure between those ingesting drinking water containing barium at 7.3 mg/L as compared to 0.1 mg/L. A concentration of 7.3 mg/L corresponds to a dose of 0.20 mg/kg/day (assuming a 70kg adult drinks two L per day).

H-2

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TABLE H-1

Summary of Toxicological Information for
Arsenic

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (proposed)	0.050
EPA MCLG (proposed)	0.050
EPA Water Quality Criteria	
fish and drinking water	2.20×10^{-6}
fish only	0.040
protection of aquatic life	0.190
EPA Drinking Water Health Advisories	
1 day	0.05 (10 kg)
10 days	0.05 (10 kg)
chronic	0.05 (10 kg) 0.05 (70 kg)
OSHA 8 hr TWA	0.01 mg/m^3
ACGIH 8 hr TWA	0.2 mg/m^3

Noncarcinogenic effects

risk characterization	
oral	
AIC	none
AIS	none
ADI	0.54 mg/kg/day
inhalation	
AIC	none
AIS	none
ADI	none
median effective dose	
oral	1.0 mg/day
inhalation	1.0 mg/day

H.2.2 Toxic and Carcinogenic Studies

The basic mechanism of action underlying the toxic action of the barium ion is thought to be an antagonism to potassium, and barium poisoning is accompanied by severe hypokalemia (potassium constitutes an effective antidote). In clinically overt poisoning barium initially causes muscular stimulation followed by paralysis, the cause of death being respiratory paralysis. The acute oral LD_{50} in man has been estimated to be about 70 mg/kg.

No toxic effects were observed in rats administered barium in drinking water at a concentration of 5 mg/L Ba^{+2} as acetate, or at 10-250 mg/L as chloride (5,6). Chickens were found to tolerate mg/kg Ba^{+2} in their diet (7).

Chronic exposure to barium sulphate dust induces a benign pneumoconosis ("baritosis") usually accompanied by little effect on pulmonary function.

Rats injected intratracheally with radioactive particles of barium sulphate has been reported in one investigation to develop bronchogenic carcinoma (8). However, for a number of reasons these results do not provide adequate evidence of carcinogenicity.

When making a risk analysis related to low concentrations of barium, due consideration should be taken of the fact that barium accompanies calcium in virtually every foodstuff (the content of barium is especially high in brazil nuts). The average daily intake has been estimated to be as high as about 1 mg/day (9). As to the presence of barium in soils, the bioavailability of barium is another important factor to take into account, especially if the contamination is due to non-toxic sulphate.

H.2.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for barium are summarized in Table H-2. The ambient water quality criterion for the protection of fresh water life is <50 mg/L. A proposed maximum concentration level in drinking water (MCL) has been established at 1 mg/L for barium. An MCLG (goal) has been proposed by the EPA at 1.5 mg/L for barium. A tentative acceptable intake for chronic exposure for both oral and inhalation routes for the non-carcinogenic effects of barium have been established at 0.5 and 1.4×10^{-4} mg/kg/day, respectively. As previously noted, barium is not considered as a carcinogen according to EPA.

TABLE H-2

Summary of Toxicological Information for
Barium

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (Proposed)	1
EPA MCLG (Proposed)	1.5
EPA Water Quality Criteria Clean Water Act Water Quality Regulation	
fish and drinking water	1.0
fish only	none
protection of aquatic life	<50
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	1.80
OSHA 8 hr TWA	$0.5 \ mg/m^3$
ACGIH 8 hr TWA	$0.5 \ mg/m^3$

Noncarcinogenic effects

risk characterization	
oral	
AIC	5.10×10^{-2} mg/kg/day
AIS	none
ADI	none
inhalation	
AIC	1.40×10^{-4} mg/kg/day
AIS	1.43×10^{-3} mg/kg/day
ADI	none
median effective dose	
oral	4.90 mg/day
inhalation	4.90 mg/day

Carcinogenic effects

Potency Factor (10^{-6} cancer risk)	none
oral	none
inhalation	none
10% effective dose	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m^3 (risk)	none
water (10^{-6} risk)	none
Classification, EPA	noncarcinogen
Classification, IARC	3

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The national interim primary drinking water standard for barium is set at 1 mg/L, which should provide for an adequate margin of safety (10).

H.3   Benzene (Gilbert et al.1980)

H.3.1   Summary of Health Effects Data

Benezene is a recognized human carcinogen. Several epidemiological studies provide sufficient evidence of a causal relationship between benzene exposure and leukemia in humans. Benzene is a known inducer of aplastic anemia in humans, with a latent period of up to 10 years. It produces leukopenia and thrombocytopenia, which may progress to pancytopenia. Similar adverse effects on the blood cell-producing system occur in animals exposed to benzene. In both humans and animals, benzene exposure is associated with chromosomal damage, although it is not mutagenic in microorganisms. Benzene was fetotoxic and caused embryolethality in experimental animals.

Exposure to very high concentrations of benzene [about 20,000 ppm $(66,000$ mg/m$^3)$ in air] can be fatal within minutes. The prominent signs are central nervous system depression and convulsions, with death usually following as a consequence of cardiovascular collapse. Milder exposures can produce vertigo, drowsiness, headache, nausea, and eventually unconsciousness if exposure continues. Deaths from cardiac sensitization and cardiac arrhythmias have also been reported after exposure to unknown concentrations. Although most benzene hazards are associated with inhalation exposure, dermal adsorption of liquid benzene may occur, and prolonged or repeated skin contact may produce blistering, erythema, and a dry, scaly dermatitis.

H.3.2   Toxic and Carcinogenic Studies

Acute exposure to high levels of benzene produce primarily central nervous system effects such as: dizziness, nausea, headaches and at very high levels (25000 ppm in air), comas and death. Lower levels of benzene do not elicit these effects no matter how long the exposure. Benzene at concentrations as low as 10 ppm can be toxic to the bone marrow and result in aplastic anemia. Benzene causes myeloblastic leukemia, acute myelomonocytic leukemia and erthryoleukemia. Immune system depression by benzene is known and more susceptibility to tuberculosis and pneumonia is seen.

No strong evidence suggests that benzene is teratogenic, however, it is a potent inhibitor of growth in utero in animals. Benzene was found non-mutagenic in several assays, but some positive

TABLE H-3

Summary of Toxicological Information for
Benzene

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (Proposed)	0.005
EPA Water Quality Criteria	
Clean Water Act Water Quality Regulation	
fish and drinking water	6.6×10^{-4}
fish only	0.040
protection of aquatic life	<5.300
EPA Drinking Water Health Advisories	
1 day	0.233 (10 kg)
10 days	0.233 (10 kg)
chronic	none
OSHA 8 hr TWA	10 ppm (30 mg/m^3)
ACGIH 8 hr TWA	10 ppm (30 mg/m^3)
Noncarcinogenic effects	
risk characterization	
oral	
AIS	none
AIC	none
ADI	none
inhalation	
AIS	none
AIC	none
ADI	none
median effective dose	
oral	8.55×10^1 mg/day
inhalation	1.70 mg/day
Carcinogenic effects	
EPA Potency Factor (10^{-6} cancer risk)	
oral	5.20×10^{-2} $(\text{mg/kg/day})^{-1}$
inhalation	2.60×10^{-2} $(\text{mg/kg/day})^{-1}$
10% effective dose	
oral	3.70×10^0 mg/kg/day
inhalation	3.70×10^0 mg/kg/day
Cancer Risk	
Inhalation at 1 ug/m^3 (risk)	4.1×10^{-6}
water (10^{-6} risk)	6.6×10^{-4}
Classification, EPA	Human carcinogen (Group A)
Classification, IARC	Human carcinogen (Class 1)

results were found in sister chromatid exchange experiments in mice.

The CAG has evaluated benzene among the fifty-four chemicals for relative carcinogenic potency as suspected human carcinogens. The level of evidence for benzene's carcinogenicity is sufficient in both animal and human studies. Based on IARC criteria, benzene is classified as a Group 1A ("known" human) carcinogen.

H.3.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for benzene are summarized in Table H-3. The ambient water quality criterion for the protection of fresh water life is <5300 ug/l. A proposed MCL has been established at 5 ug/L for benzene. Regulations for workplace exposure to benzene have been established by OSHA at 10 ppm (30 mg/m^3) and by ACGIH at 10 ppm (30 mg/m^3).

H.4 Toluene (USEPA 1985a, USEPA 1985b

H.4.1 Summary of Health Effects Data

There is no conclusive evidence that toluene is carcinogenic or mutagenic in animals or humans. Oral administration of toluene at doses as low as 260 mg/kg produced a significant increase in embryotoxic lethality in mice. Decreased fetal weight was observed at doses as low as 434 mg/kg, and increased incidence of cleft palate was seen at doses as low as 867 mg/kg. However, other researchers have reported that toluene is embryotoxic but not teratogenic in laboratory animals. Acute exposure to toluene produces central nervous system depression and narcosis in humans. However, even exposure to quantities sufficient to produce unconsciousness fail to produce residual organ damage. Chronic inhalation exposure to toluene at relatively high concentrations produces cerebellar degeneration and an irreversible encephalopathy in mammals. The oral LD_{50} value and inhalation LC_{LO} value for the rat are 5,000 mg/kg and 15,000 mg/m^3, respectively.

H.4.2 Toxic and Carcinogenic Studies

The compound, toluene, does not appear to present carcinogenic risks based upon data available at this time. Exposures to levels of toluene did not produce an increased incidence of neoplastic, proliferative, inflammatory, or degenerative lesions in rats. Toluene does not appear to be carcinogenic when applied to the shaved skin of mice. Toluene has been tested for genotoxicity (mutagenicity) by many investigators using various assay methods and has not been demonstrated to be genotoxic.

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Investigations into the teratogenic (reproductive effects) of toluene on mice resulted in statistically significant increases in the incidence of cleft palate. However, no studies have been conducted in humans. The acceptable intakes for chronic and subchronic exposure via the oral route are 3.00×10^{-1} and 4.30×10^{-1} mg/kg/day, respectively. The acceptable intake for both chronic and subchronic exposure via the inhalation route is 1.50×10^{0} mg/kg/day.

H.4.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for toluene are summarized in Table H-4. The ambient water quality criterion for the protection of fresh water life is <17.5 mg/L. The acceptable intake for chronic exposure via inhalation and oral routes are 1.50×10^{0} and 3.00×10^{-1} mg/kg/day, respectively. The acceptable intakes for subchronic exposure via inhalation and oral routes are 1.50×10^{0} and 4.30×10^{-1} mg/kg/day, respectively. Regulations for workplace exposure developed by OSHA and ACGIH are 375 mg/m^{3}. As previously noted, toluene is considered to be a non-carcinogen by EPA.

H.5 Trichloroethylene (US EPA, 1985a,b,c)

H.5.1 Summary of Health Effects Data

Trichloroethylene (TCE) has a low acute toxicity with an acute oral LD$_{50}$ value in several species ranging from 6000-7000 mg/kg. Chronic exposure in rodents have been found to cause adverse effects on liver and kidneys at high doses. In long-term studies TCE has induced hepatocellular carcinomas in mice. Due to presence of carcinogenic impurities in the test compounds and other factors, the significance of these findings are not clear. Extensive epidemiological investigations have failed to substantiate an increased carcinogenic risk in man. Also, results from short-term testing have been ambiguous.

H.5.2 Toxic and Carcinogenic Effects

TCE has a low acute toxicity in mammals. In man higher concentrations of this volatile substance has anesthetic and analgesic properties and is known to occasionally elicit cardiac arrythmias. Chronic exposure has been reported to induce neurotoxic symptoms like ataxia, sleep disturbances and psychotic episodes as well as trigeminal neuropathy.

In rodents TCE causes toxic effects to the kidney tubuli and liver. No significant signs of developmental toxicity has been found in inhalation experiments using these experimental animals.

H-6

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TABLE H-4

Summary of Toxicological Information for
Toluene

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCLG (Proposed)	2
EPA Water Quality Criteria	
Clean Water Act Water Quality Regulation	
fish and drinking water	1.43×10^1
fish only	4.24×10^2
protection of aquatic life	<17.5
EPA Drinking Water Health Advisories	
1 day	18 (10 kg)
10 days	6 (10 kg)
chronic	10.8
OSHA 8 hr TWA	100 ppm (375 mg/m^3)
ACGIH 8 hr TWA	100 ppm (375 mg/m^3)

Noncarcinogenic effects

risk characterization	
oral	
AIC	3.00×10^{-1} mg/kg/day
AIS	4.30×10^{-1} mg/kg/day
ADI	none
inhalation	
AIC	1.50×10^0 mg/kg/day
AIS	1.50×10^0 mg/kg/day
ADI	none
median effective dose	
oral	2.69×10^3 mg/day
inhalation	2.69×10^3 mg/day

Carcinogenic effects

Potency Factor (10^{-6} cancer risk)	none
oral	none
inhalation	none
10% effective dose	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m^3 (risk)	none
water (10^{-6} risk)	none
Classification, EPA	noncarcinogen
Classification, IARC	3

Several long-term studies in rodents have been negative as to carcinogenic effects, but an unequivocal increase of the incidence of liver tumors has been found in B6C3F$_1$ mice (NTP). An increase of renal tubular-cell tumors was also observed in male F344 rats, but this assay (NTP) has been considered inadequate to evaluate the carcinogenic response. As to the results of short-term tests the outcome has been variable. According to EPA a mutagenic potential cannot be ruled out, but if the compound is mutagenic, the available data suggest that TCE would be a very weak, indirect mutagen. The purity of TCE, used in may of the reported studies, represent a key confounding factor (epichlorohydrin or 1,2-epoxy-butane are often added as stabilizers).

On the basis of the long-term studies in rodents EPA has classified TCE as a Group B2 carcinogen. EPA's position as to carcinogen classification of TCE and a few other halogenated hydrocarbons has been challenged by representatives from the scientific community and industry, the major thrust of the criticism being the interpretation of the significance of the mouse liver tumors found. It has been argued that an increase in the incidence of these tumors - which is already high in controls of this particular strain of mice - may be caused by a toxic action on the liver at the high doses of TCE used in these bioassays rather than due to a carcinogenic action of the chemical per se. The use of the linearized multistage model for calculation of carcinogenic potency, which is of the same order as that for benzene or vinyl chloride, has also been questioned. If TCE mainly acts as a promoter this value is certainly far to high. IARC considers that only limited evidence is available that TCE is carcinogenic in mice and has classified the substance in Group 3, a chemical which cannot be classified as to its carcinogenicity for humans.

H.5.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for TCE are summarized in Table H-5. The ambient water quality criterion for the protection of freshwater life is <45,000 ug/L. A proposed maximum concentration level in drinking water (MCL) has been established at 5 ug/L for TCE. Regulations for workplace exposures have been developed by OSHA (100 ppm TWA or 540 mg/m^3) and ACGIH (50 ppm or 270 mg/m^3).

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TABLE H-5

Summary of Toxicological Information for
Trichloroethene

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (proposed)	0.005
EPA MCLG (proposed)	0
EPA Water Quality Criteria	
fish and drinking water	2.7×10^{-4}
fish only	8.07×10^{-2}
protection of aquatic life	45.0
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA	100 ppm (540 mg/m^3)
ACGIH 8 hr TWA	50 ppm (270 mg/m^3)

Noncarcinogenic effects

risk characterization	
oral	none
AIC	none
AIS	none
ADI	0.54 mg/kg/day
inhalation	
AIC	none
AIS	none
ADI	none
median effective dose	
oral	9.5 mg/day
inhalation	1.05 mg/day

Carcinogenic effects

EPA Carcinogenic Potency Factor (10^{-6} risk)	
oral	1.0×10^{-2} (mg/kg/day)$^{-1}$
inhalation	4.63×10^{-3} (mg/kg/day)$^{-1}$
10% effective dose	
oral	5.56 mg/kg/day
inhalation	5.56 mg/kg/day
Cancer Risk	
Inhalation at 1 ug/m^3	4.1×10^{-6}
water (10^{-6} risk)	2.7 ug/L
Classification, EPA	B2
Classification, IARC	insufficient evidence

H.6 1,2,4-Trichlorobenzene (USEPA October 1985)

H.6.1 Summary of Health Effects Data

There are no reports indicating carcinogenic, teratogenic, or
mutagenic activity of the trichlorobenzenes in humans or animals.
No specific reproductive effects have been found for the
trichlorobenzenes, but embryotoxicity has been noted at a dose
level that produces maternal toxicity in rats.

Several animal studies on the subchronic toxicity to
trichlorobenzene have been reported. Inhalation studies with
1,2,4-trichlorbenzene of 1.5 to 6 months duration in rats,
rabbits, dogs, and monkeys have not shown major irreversible
effects, although some effects on liver and kidney were found
(transient histological changes and increased relative liver
weight). Increased urinary porphyrin levels were also noted. A
study reported that mice exposed to trichlorobenzene (isomers
unspecified) for 3 weeks to 3 months showed indications of bone
marrow damage. In a chronic study in which mice were
administered 1,2,4-triclorobenzene by dermal application, there
was a treatment-related increase in the incidence of amyloidosis,
which affected a number of organs and was considered a primary
cause of death.

TCB is an inducer of the microsomal mixed function oxidases and
therefore will increase metabolism, leading to the inactivation
or activation of chemicals affected by this system.

H.6.2 Toxic and Carcinogenic Data

Human exposure to 1,2,4-trichlorobenzene at 3-5 ppm causes eye
and respiratory irritation. The only other data on human
exposure are individual case reports of aplastic anemia of
persons exposed occupationally or domestically. The effect in
mammals of acute exposures by various routes to trichlorobenzenes
include local irritation, convulsions, and death. Limited
studies indicate negative effects towards mutagenic, teratogenic
and carcinogenic effects caused by exposure to
1,2,4-tirchlorobenzene. EPA has classified this compound as a
noncarcinogen and the IARC has classified 1,2,4-trichlorobenzene
as a Group 3.

H.6.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for
1,2,4-trichlorobenzene are summarized in Table H-6. The ambient
water quality criterion for the protection of freshwater life is
<0.250 mg/L. To date, standards for the protection of human
health due to drinking water and food has not been established
for 1,2,4-trichlorobenzene. An acceptable intake for chronic

exposure to this compound via the oral route is 2.0×10^{-2} mg/kg/day. Regulations for work place exposure developed by ACGIH is 40 mg/m^3. As previously noted, 1,2,4-trichlorobenzene is considered to be a noncarcinogen by EPA.

H.7   Chloroform (US EPA,1985 and Perwak et al.1980)

H.7.1   Summary of Health Effects Data

Chloroform (trichloromethane) is produced during the chlorination of drinking water and thus is a common drinking water contaminant. Chronic administration of chloroform by gavage is reported to produce a dose-related increase in the incidence of kidney epithelial tumors in rats and a dose-related increase in the incidence hepatocellular carcinomas in mice. Epidemiological studies suggest that higher concentrations of chloroform and other trihalomethanes in water supplies may be associated with the increased frequency of bladder cancer in humans. However, these results are not sufficient to establish causality. An increased incidence of fetal abnormalities in the offspring of pregnant rats exposed to chloroform by inhalation has been observed. Oral doses of chloroform that cause maternal toxicity produce relatively mild fetal toxicity in the form of reduced birth weight. There are limited data suggesting that chloroform has mutagenic activity in some test systems. However, negative results have been reported for bacterial mutagenesis assays.

Humans may be exposed to chloroform by inhalation, ingestion, or skin contact. Toxic effects include local irritation of the skin or eyes, central nervous system depression, gastrointestinal irritation, liver and kidney damage, cardiac arrhythmia, ventricular tachycardia, and bradycardia. Death from chloroform overdose can occur and is attributed to ventricular fibrillation. Chloroform anesthesia can produce delayed death as a result of liver necrosis. Exposure to chloroform by inhalation, intragastric administration, or intraperitoneal injection produced liver and kidney damage in laboratory animals. The oral LD$_{50}$ and inhalation LC$_{LO}$ values for the rat are 908 mg/kg and 39,000 mg/m^3 for four hours, respectively.

H.7.2   Toxic and Carcinogenic Studies

Chloroform is among the fifty-four chemicals evaluated by CAG for relative carcinogenic potencies as suspected human carcinogens. A level-of-evidence in animals indicates that sufficient studies have been conducted to determine the carcinogenicity of chloroform. However, inadequate studies have been conducted to determine the level of carcinogenic evidence in humans. Therefore, IARC has ranked chloroform as a B2 ("probable" human

TABLE H-6

Summary of Toxicological Information for
1,2,4-Trichlorobenzene

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (proposed)	none
EPA MCLG (proposed)	
EPA Water Quality Criteria	
fish and drinking water	insufficient data
fish only	insufficient data
protection of aquatic life	<0.250
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA	none
ACGIH 8 hr TWA	40 mg/m^3

Noncarcinogenic effects

risk characterization	
oral	
AIC	2.0 x 10^{-2} mg/kg/day
AIS	none
ADI	none
inhalation	
AIC	none
AIS	none
ADI	none
median effective dose	
oral	3.73 x 10^1 mg/day
inhalation	1.32 x 10^1 mg/day

Carcinogenic effects

Potency Factor (10^{-6} risk)	none
oral	none
inhalation	none
10% effective dose	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m^3	none
water (10^{-6} risk)	none
Classification, EPA	noncarcinogen
Classification, IARC	3

carcinogen) compound based upon the level of evidence in animal studies.

H.7.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for chloroform are summarized in Table H-9. The ambient water quality criterion for the protection of fresh water life is <28,900 ug/L. A MCL has been established at 100 ug/L for trihalomethanes (chloroform). Regulations for workplace exposure are 50 ppm (240 mg/m^3) for OSHA and 10 ppm (50 mg/m^3) for ACGIH.

H.8 Xylene (ENVIRON, 1987)

H.8.1 Summary of Health Effects Data

Acute exposure to high levels of xylene (1,2- 1,3- or 1,4-xylene) produces CNS depression. Chronic exposure to xylene has produced equivocal hematological effects in experimental animals. Xylene has been shown to cause adverse reproductive effects in animals.

ENVIRON has estimated an acute Acceptable Daily Intake (ADI) for toluene based on a USEPA (1985) one-day health advisory and subchronic and chronic ADI's based on an inhalation NOAEL from a reproductive study in rats.

H.8.2 Toxic and Carcinogenic Effects

Acute Exposure

When inhaled at high concentrations, xylene causes CNS depression. High concentrations can also cause reddening of the face, disturbed vision, and salivation. There is some evidence suggesting that xylene sensitizes the myocardium to the endogenous neurohormone, epinephrine, and can precipitate heart failure and death. The American Conference of Governmental Industrial Hygienists has recommended a TLV for xylene of 100 ppm.

USEPA (1985) has established a "one-day health advisory" for xylene of 12 mg/l in drinking water for a 10 kg child. This one-day level is based on an experiment conducted in human subjects by Gamberale et al. (1978) in which an inhalation concentration of 1,300 mg/m^3 for one hour caused CNs effects. ENVIRON has calculated an acute ADI for xylene based on this study, as follows: (1300 mg/m^3) x (1 m^3/1000 l) x (29 l/min) x (60 min/day) x (1/70 kg) = 32.3 mg/kg/day.

TABLE H-7

Summary of Toxicological Information for
Chloroform

Relevant Requirements, Criteria, Advisories or Guidance	Value
EPA MCL (trihalomethanes)	100 ug/L
EPA Water Quality Criteria	
fish and drinking water	1.9×10^{-1} ug/L
fish only	15.7 ug/L
protection of aquatic life	<28900 ug/L
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA	50 ppm (240 mg/m^3)
ACGIH 8 hr TWA	10 ppm (50 mg/m^3)

Noncarcinogenic effects

risk characterization	
oral	none
AIC	none
median effective dose	none
oral	none
inhalation	none

Carcinogenic effects

EPA Potency Factor (10^{-6} cancer risk)	8.1×10^{-2} (mg/kg/day)$^{-1}$
oral	7.00×10^{-2} (mg/kg/day)$^{-1}$
inhalation	none
10% effective dose	
oral	5.00×10^{-1} mg/kg/day
inhalation	5.00×10^{-1} mg/kg/day
Cancer Risk	
Inhalation at 1 ug/m^3 (risk)	2.3×10^{-6}
water (10^{-6} risk)	1.9×10^{-1}
Classification, EPA	Group B2
Classification, IARC	Group 2B

Subchronic Exposure

There have been several recent studies which indicate that xylene exposure causes adverse reproductive and teratogenic effects in animals. A study by Mirkova et al. (1983), who reported that xylene caused embryotoxic effects in rats after inhalation exposure, has been used as the basis for the subchronic ADI. In this study, rats were exposed to xylene at a concentration of 10, 50, or 500 mg/m^3, 6 hr/day, 5 days/wk from days 1 through 21 of gestation. Significant embryotixic effects were produces after exposure to the two higher concentrations but not to the lowest concentration. Using the NOAEL of 10 mg/m^3 and a safety factor of 100, a subchronic ADI of 0.0103 mg/kg/day was calculated, as follows: (10 mg/m^3) x (1 m^3/1000 l) x (0.1 l/min) x (360 min/day) x (5 days/7 days) x 1/0.25 kg) x (1/100) = 0.0103 mg/kg/day.

Chronic Exposure

Workers chronically exposed to xylene occupationally display symptoms similar to those seen in acutely exposed indivuduals (Sandmeyer 1981). In addition, there have been reports that disturbances in the blood can occur from xylene exposure. These effects, however, may be due to benzene contamination. No data on the potential carcinogenicity or mutagenicity of xylene were available in the literature.

ENVIRON has calculated a chronic ADI for xylene based on the reporductive toxicity study by Mirkova et al. (1983), described above, in which xylene caused embryotixic effects in rats following exposure by inhalation. Using the NOAEL of 10 mg/m^3 and a safety factor of 1,000, a chronic ADI of 0.00103 mg/kg/day was calculated, as follows: (10 mg/m^3) x (1 m^3/1000 l) x (0.1 l/min) x (360 min/day) x (5 days/7days) x (1/0.25 kg) x (1/1000) = 0.00103 mg/kg/day.

H.8.3   Applicable and Relevant Standards

The recognized applicable and relevant standards for xylene are summarized in Table H-8. The ambient water quality criterion for the protection of freshwater life has not been established. A proposed MCLG has been established at 0.44 mg/L, for xylene. Regulations for workplace exposures have been developed by OSHA (100 ppm TWA or 435 mg/m^3) and ACGHI (100 ppm or 435 mg/m^3). As previously noted, xylene is considered to be a noncarcinogen.

TABLE H-8

Summary of Toxicological Information for
Xylene

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (proposed)	
EPA MCLG (proposed)	0.44
EPA Water Quality Criteria	
fish and drinking water	none
fish only	none
protection of aquatic life	none
EPA Drinking Water Health Advisories	
1 day	12
10 days	none
chronic	none
OSHA 8 hr TWA	100 ppm (435 mg/m^3)
ACGIH 8 hr TWA	100 ppm (435 mg/m^3)

Noncarcinogenic effects

risk characterization	
oral	
AIC	0.01 mg/kg/day
AIS	0.1 mg/kg/day
ADI	0.54 mg/kg/day (EPA
inhalation	32.3 mg/kg/day (ENVIRON)
AIC	0.4 mg/kg/day
AIS	0.69 mg/kg/day
ADI	none
median effective dose	
oral	none
inhalation	none

Carcinogenic effects

Carcinogenic Potency Factor (10^{-6} risk)	
oral	none
inhalation	none
10% effective dose	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m^3	none
water (10^{-6} risk)	none
Classification, EPA	NC
Classification, IARC	3

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H.9 1,2,3-Trichloropropane (ENVIRON, 1987)

H.9.1 Summary of Health Effects Data

Short-term exposure in humans to 1,2,3-trichloropropane (1,2,3-TCP) has produced eye and throat irritation. In animals, central nervous system depression, liver damage, and eye irritation have been reported as a result of acute exposure. Subchronic exposure in animals has led to liver, kidney, and nasal turbinate changes, altered hematological parameters and reduced pseudocholinesterase levels.

H.9.2 Toxic and Carcinogenic Effects

Acute Exposure

Acute inhalation exposure to 1,2,3-TCP in humans has produced eye and throat irritation and an unpleasant odor at 100 ppm (Silverman et al. 1946). ACGIH (1986) describes that a "borderline majority found that a concentration of 50 ppm was acceptable for an 8-hour workday". The conditions defining acceptability were not reported by ACGIH (1986).

Animals exposed to high concentrations of 1,2,3-TCP have experienced central nervous system depression, liver damage, and eye irritation. Rats and guinea pigs exposed to 800 ppm for 30 minutes experienced central nervous system depression. Higher levels produced narcosis and convulsions (Lewis 1979). Mice exposed for 20 minutes to 5,000 ppm died several days later from liver damage (McOmie and Barnes 1949). Smyth and coworkers (1962) report that 1,2,3-TCP is highly irritating to the eyes of rabbits. Rats and mice exposed to 125 ppm for four hours experienced breathing difficulties, inactivity, and eye and nasal irritation (Dow 1985). Higher levels caused mortality in all animals. Rats exposed to 0.35 or 0,8 mg/L for 7 days developed histopathological changes in the liver (Bonashevskays and Belyaeva 1975). Fat cell degeneration and disintegration were seen in rats exposed to 0.35 mg/L for 7 days (Tarasova et al. 1977). Mast cell changes were also reported in rats exposed to 0.4 mg/L for 7 days (Tarasova 1975).

ACGIH (1986) recommends a TLV-TWA of 10 ppm (60 mg/m^3) to protect occupationally exposed persons from adverse effects due to long-term exposure. The basis for the TLV-TWA is the NTP subchronic study in rats. ACGIH assumed the NOAEL was 8.0 mg/kg/day. Assuming a 70 kg man and a breathing rate of 10 m^3/day, this can be converted to 56 mg/m^3 which is approximately 10 ppm. An acute 24-hour limit for environmental exposures in humans suggested by ENVIRON is 8.0 mg/kg/day based on the TLV-TWA which in turn is based on the subchronic rat gavage study

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conducted by NTP (1983). The oral data is used since adequate inhalation data are not available.

Subchronic Exposure

In a four-month gavage study in rats conducted for NTP (Hazelton 1983a), rats received doses of 0, 8, 16, 32, 63, 125, or 250 mg/kg body weight, 5 days/week, for 120 weeks. All rats in the high-dose group died within five weeks. Rats exposed to doses of 63 mg/kg and higher experienced weight loss and clinical signs including thinness, hunched appearance, depression, and abnormal eye and urine stains.

Liver absolute and relative weights were increased in the 32, 63, and 125 mg/kg groups as well as in the 16 mg/kg female rats. Kidney absolute and relative weights were increased in the rats receiving doses of 32 mg/kg or higher. A statistically dimished testes-to body weight ratio without histopathological alterations was reported in the 125 mg/kg males.

Hematological parameters including hematocrit, hemoglobin, and erythrocyte counts were reduced in dose groups of 16 mg/kg and higher, although the changes appearing in the 16 and 32 mg/kg groups were described as minimal.

Hematological parameters including hematocrit, hemoglobin, and erythrocyte counts were reduced in dose groups of 16 mg/kg and higher, although the changes appeaing in the 16 and 32 mg/kg groups were described as minimal.

Pseudocholinesterase levels were depressed in males exposed to 63 mg/kg and higher and females treated with 16 mg/kg or more. Diminished blood urea nitrogen levels were reported among females receiving 32 mg/kg or more.

Histopathological examination revealed liver and kidney changes and inflammation and necrosis of nasal turbinates. Liver changes including necrosis, bile duct inflammation, and hyperplasia were not evident below the 125 mg/kg dose level. Kidney alterations consisting of hyperplasia and chronic progressive nephropathy were seen in rats exposed to 63 mg/kg and higher.

The responses of B6C3F$_1$ mice exposed to 1,2,3-TCP under a similar paradigm resembled those seen in rats (Hazelton 1983b). However, the effects were seen at levels slightly higher than those reported for rats. In addition, depressed sperm counts occurred at 125 mg/kg and 250 mg/kg.

The rats were the most sensitive species tested by NTP (Hazelton 1983a,b) and the NOEL identified from this study is 8 mg/kg, 5 days/week, which translates into 5.7 mg/kg/day. The NOEL is used

with a 100-fold safety factor to estimate a subchronic ADI of 0.057 mg/kg/day. Although a lower NOEL could be derived from a Russian inhalation study, the validity of the study is questionable, and we rely on the NTP study (Hazelton 1983a) for risk assessment.

Chronic Exposure

Data pertaining to chronic exposure in humans or animals are not available at this time. A chronic ADI can be estimated from the oral subchronic study conducted by Hazelton (1983a) discussed in the previous section by applying a safety factor of 1000. Thus, the chronic ADI is estimated to be 0.0057 mg/kg/day.

H.9.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for 1,2,3-trichloropropane are summarized in Table H-9. The ambient water quality criterion for the protection of freshwater life has not been established. A proposed MCL and MCLG have not been established. Regulations for workplace exposures have been developed by OSHA and ACGIH at 50 ppm or 300 mg/m^3 1,2,3-Trichloropropane is considered to be a probable human carcinogen by EPA.

H.10 Tetrachloroethylene (ENVIRON, 1987)

H.10.1 Summary of Health Effects Data

Tetrachloroethylene (PCE) is carcinogenic is animals. Effects in humans include central nervous system depression, liver damage, and skin irritation. Effects in animals include central nervous system depression; liver and kidney damage; behavioral effects; and mucous membrane irritation. Tetrachloroethylene itself is only a week mutagen in microbial test systems, however, its expoxide intermediate is mutagenic.

H.10.2 Toxic and Carcinogenic Effects

Acute Exposure

Acute exposure to PCe in humans can result in liver damage and central nervous system depression. Impaired liver function, as demonstrated by altered liver enzyme activity, increased urinary urobilinogen, and increased total serum bilirubin has been reported in workers exposed to PCE (Stewart et al. 1961, Hake and Stewart 1977). Central nervous system effects have also been reported (Stewart et al. 1970, Rowe et al. 1952, Stewart et al. 1977). Symptoms including dizziness, confusion, headache, nausea, speech difficulty and numbness occurred when humans were

TABLE H-9

Summary of Toxicological Information for
1,2,3-Trichloropropane

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (proposed)	none
EPA MCLG (proposed)	none
EPA Water Quality Criteria	
fish and drinking water	none
fish only	none
protection of aquatic life	none
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA	50 ppm (300 mg/m^3
ACGIH 8 hr TWA	50 ppm (300 mg/m^3)

Noncarcinogenic effects

risk characterization	
oral	
AIC	none
AIS	none
ADI	none
inhalation	
AIC	none
AIS	none
ADI	none
median effective dose	
oral	none
inhalation	none

Carcinogenic effects

EPA Carcinogenic Potency Factor	
oral	0.1 $(mg/kg/day)^{-1}$
inhalation	0.1 $(mg/kg/day)^{-1}$
10% effective dose	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m^3	none
water (10^{-6} risk)	none
Classification, EPA	B2
Classification, IARC	3

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exposed to between 100 and 200 ppm (678 and 1356 mg/m^3) PCE for 10 minutes to 2 hours. Tetrachloroethylene is a skin irritant (Hake and Stewart 1977). Exposure to high but unknown concentrations may also cause lung damage (Levine et al. 1981). A case report by Abedin et al. (1980) suggests that premature ventricular beats may be attributable to PCE exposure. The ambient levels were not reported although the PCE plasma level of the patient was 3.8 ppm.

An extensive review of the effects of acute exposure of animals to tetrachloroethylene has been provided by USEPA (1985). The results highlighted in the following discussion include some of the more outstanding findings. Effects in animals due to inhalation of tetrachloroethylene have included anesthesia, liver and kidney toxicity, behavioral effects, heart effects, and primary skin and eye irritation. Anesthesia has occurred in rats at vapor concentrations of 2500 ppm (16,950 mg/m^3) for 16 hours (Carpenter 1937, Rowe et al. 1952). Other central nervous system effects have included behavioral effects such as increased salivation, reflexive aggression in rats (10,848 mg/m^3, 7 hr/day, 18 days) (Rowe et al. 1952), reduced escape and avoidance response in rats (15,594 mg/m^3, 4 hr/day, 5 days/wk, 2 weeks) (Goldbert et al. 1964), and alterned preening rate and time (200 ppm, 6 hr/day, 4 days), (Savolainen et al. 1977). Tetrachloroethylene apparently sensitizes the heart to epinephrine as demonstrated by ventricular arrhythmias, premature contractions, and tachycardia in rabbits and dogs (Kobayashi et al. 1982).

ACGIH (1986) suggests a TLV-TWA of 50 ppm (340 (mg/m^3) for workers exposed to PCE. This is intended to prevent discomfort and subjective complaints which might occur at levels from 100 to 200 ppm. The TLV-TWA can be expressed as 67.6 mg/kg/day, assuming a human breathing rate of 29 L/minute, body weight of 70 kg, and an exposure duration of 8 hours/day. An acute 24-hour inhalation limit for environmental exposure to humans, suggested by ENVIRON, is 67.6 mg/kg/day.

Subchronic Exposure

The effects of extended exposure to tetrachloroethylene in humans are primarily in the liver. Altered liver function (Hughes 1954), cirrhosis (Coler and Rossmiler 1953), hepatitis (Hughes 1954), cell necrosis, and liver enlargement (Meckler and Phelps 1966) occurred at high or unknown exposure levels. Many of the same symptoms seen in acute exposures are evident in longer-term exposures. In addition, long-term exposure has been associated with short-term memory deficits, ataxia, irritability, disorientation, sleep disturbances, and decreased alcohol tolerance (Stewart et al. 1977).

Tetrachloroethylene has produced liver and kidney effects upon subchronic exposure in animals. Effects on the liver of animals include congestion and swelling in rats (3188 mg/m^3 or 1561 mg/m^3, 8 hr/day, 5 day/wk 7 months) (Carpenter 1937), increased liver weights, fatty degeneration, and cirrhosis in guinea pigs (LOEL was 678 mg/m^3, 7 hr/day, 5 days/wk, 6 months) (Rowe et al. 1952). Kidney damage was evident by cloudy swelling, desquamation, congestion (Carpenter 1937), increased kidney weight (Rowe et al. 1952), decreased glomerular filtration and increased tubular excretion (Brancaccio et al. 1971).

Behavioral effects have been reported and they include ataxia, somnolence, and anesthesia in rats exposed to relatively high concentrations (1150 ppm to 2300 ppm) (Row et al. 1952, Goldbert et al. 1964). At 200 ppm, preening rate and time were slightly affected (Savolainen et al. 1977).

Available data thus far do not indicate any significant reproductive or teratogenic effects due to tetrachloroethylene exposure, according to USEPA (1985). USEPA (1985) claims that any anatomical alterations seen in tetratogenicity studies can be considered to be reversible. However, it cannot be ascertained from the study results whether the effects actually are reversible, because most study protocols apparently did not allow for a reversibility period upon cessation of exposure. Also, it is possible that the reversible effects in one species may be more severe in another species. Minor behavioral effects were attributed to maternal toxicity (USEPA 1985).

Schwetz and coworkers (1975) exposed rats and mice to 300 ppm (2034 mg/m^3) for 7 hr/day on days 6 to 15 of gestation. Increases in the number of runts, subcutaneous edema, and delayed ossificiation of the skull and sternebrae were reported in mice, although the increases in incidence were not statistically significant. In rats, there was a slight but statistically significant increase in resporption, but no malformations or other reproductive effects were reported.

Nelson and colleauges (1980) exposed pregnant rats to 900 ppm (6102 mg/m^3) on days 7 to 13 to 14 to 20 of gestation. Slight, but reversible, behavioral effects in offspring were evident by decreased performance in motor activity tests at doses for which maternal toxicity was apparent.

Embryotoxicity was reported to occur when rats were exposed to 1000 ppm PCE prior to mating (Tepe et al. 1982). However, no effects were seen in the surviving offspring upon maturation (Manson et al. 1982). Beliles et al. (1980) did not find any teratogenic or reproductive effects in rabbits exposed to 500 ppm (3390 mg/m^3), 7 hr/day, 5 day/week, for three weeks pre-gestation and on days 0 to 21 of gestation. Additionally, Beliles et al.

(1980) also observed altered sperm morphology in mice but not rats.

No effects were seen at an exposure level of 475 mg/m^3 in the Carpenter (1937) study. USEPA has calculated an RfD/ADI of 0.02 mg/kg/day for tetrachloroethylene based on the kidney and liver changes seen at higher doses (USEPA 1986a). Assumptions used by USEPA (1986a) to calculate this RfD/ADI include a human breathing rate of 1 m^3/hour, an exposure of 8 hours/day, for 5 days/week, an inhalation retention factor of 1000 was then applied for inter- and intraspecies conversion and for the use of subchronic data in the derivation of a chronic exposure limit.

ENVIRON estimates a subchronic ADI of 0.65 mg/kg/day based on the NOEL 475 mg/m^3 seen in the Carpenter (1937) study. Assumptions inherent to this estimation include a rate breathing rate of 0.1 L/day, rat body weight of 0.25 kg, and an exposure for 8 hours/day, 5 days/week. A safety factor of 100 was used for inter- and intraspecies conversion. An additional factor of 10 was not used in this case since this estimation is intended to be a subchronic limit.

Chronic Exposure

Tetrachlorethylene was carcinogenic in mice but not in rats during an oral administration study conducted by the National Cancer Institute (NCI 1977). Groups of 50 mice of each sex received tetrachloroethylene in corn oil by gavage 5 days per week for 78 weeks. Time-weighted average doses were 536 or 1072 mg/kg/day and 386 or 772 mg/kg/day in males and females, respectively. The incidence of hepatocellular carcinoma was 2/20, 32/49, and 27/48 in control, low-, and high dose males, respectively. Female mice developed hepatocellular carcinoma at incidences of 2/20, 1948, and 19/48 in the control, low-, and high-dose groups, respectively.

Rats received time-weighted average doses of 471 and 941 mg/kg/day and 474 or 949 mg/kd/day in male and female groups, respectively. No increased incidences of tumors attributable to treatment were observed.

A subsequent bioassay conducted by the National Toxicology Progam (NTP 1986) investigated the carcinogenicity of tetrachloroethylene by the inhalation route of exposure in rats and mice. Animals were exosed 6 hr/day, 5 days/week, for 2 years to 0, 200, or 400 ppm (rats) and 0, 100, or 200 ppm (Mice) tetrachloroethylene. A small statistically significant increase of mononuclean cell leukemia was seen in treated male and female rats (28/50, 37/50, 37/50, and 18/50, 30/50, 29/50 in control, low, and high dose male and female rats, respectively). A dose-related, although not statistically significant, increase in

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rare kidney tumors was also observed in male rats. increased incidences of hepatocellular carcinoma developed in treated male and female mice (7/49, 25/49, 26/50 and 1/48, 13/50, 36/50 in control, low- and high-dose male and female mice, respectively).

The epoxide intermediate of tetrachloroethylene was mutagenic in bacterial studies (Kline et al. 1982) even though the parent compound itself was negative or weakly mutagenic in the presence of a metabilic activation system.

According to EPA classification, the results from oral and inhalation studies together with the mutagenicity of the epoxide intermediate provide sufficient evidence for the carcinogenicity of tetrachloroethylene in animals.

As described in the subchronic exposure section, USEPA (1986a) has estimated an RfD/ADI for chronic exposure of 0.02 mg/kg/day based on a subchronic rat study and with the intention of protecting humans from non-carcinogenic effects.

H.10.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for tetrachloroethylene are summarized in Table H-10. The ambient water quality criterion for the protection of freshwater life is <5.3 mg/L. A proposed MCLG has been established at 0 mg/L, for PCE. Regulations for workplace exposures have been developed by OSHA (100 ppm TWA or 670 mg/m^3) abd ACGIH (50 ppm or 335 mg/m^3).

H.11 Ethylbenzene (ENVIRON, 1987)

H.11.1 Summary of Health Effects Data

Acute Exposure

Ethylbenzene is absorbed through the skin, by inhalation, and by ingestion in humans. In humans, acute inhalation of 2,000-5,000 ppm causes intolerable irritation of the nose, eyes, and throat, excessive tearing (lacrimation), and dizziness. Exposure to 200 ppm causes transient irritation (Gerade 1963). According to one report, exposure to 2,000 ppm for an uspecified duration causes coordination disorders, dizziness, narcosis, and convulsions (Ivanov 1964).

In animals, adsorption also occurs by skin, inhalation, and ingestion. Acute inhalation exposure of rats to 5,000 ppm for 30-60 minutes on 10,000 ppm for 3 minutes resulted in death. Cause of death was associated with intense congestion and edema of the lungs, and generalized visceral hyperemia (Yant et al.

TABLE H-10

Summary of Toxicological Information for
Tetrachloroethylene

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (proposed)	none
EPA MCLG (proposed)	0
EPA Water Quality Criteria	
fish and drinking water	8.0×10^{-4}
fish only	8.85×10^{-3}
protection of aquatic life	<5.3
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA	100 ppm (670 mg/m^3)
ACGIH 8 hr TWA	50 ppm (335 mg/m^3)

Noncarcinogenic effects

risk characterization	
oral	
AIC	0.02 mg/kg/day
AIS	none
ADI	none
inhalation	
AIC	none
AIS	none
ADI	none
median effective dose	
oral	1.46×10^3 mg/day
inhalation	7.27×10^3 mg/day

Carcinogenic effects

EPA Carcinogenic Potency Factor	
oral	5.1×10^{-2} (mg/kg/day)$^{-1}$
inhalation	1.70×10^{-3} (mg/kg/day)$^{-1}$
10% effective dose	
oral	3.23 mg/kg/day
inhalation	3.23 mg/kg/day
Cancer Risk	
Inhalation at 1 ug/m^3	none
water (10^{-6} risk)	8.85 ug/L
Classification, EPA	B2
Classification, IARC	2

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1930). In laboratory rats, an oral LD$_{50}$ fo 3,500 mg/kg has been reported (Wolf et al. 1956).

The ACGIH has established a TLV of 100 ppm (435 mg/m^3) for ethylbenzene (ACGIH 1986). The USEPA (1985) has established a "one-day health advisory" for drinking water fo 21 mg/l for a 10 kg child. Derivation of the one-day level is based on a 100 ppm (435 mg/m3) NOAEL identified in 18 human male voluteers following a single 8-hour inhalation exposure (Bardodej and Bardodejova 1970). An inhalation adsorption efficiency of 50% was assumed, based on human and rat data (Bardodej and Bardodejova 1970; Chin et al. 1980). ENVIRON has calculated an acute ADI for ethylbenzene based on these data, as follows: (435 mg/m^3) x (1 m^3/1000 l) x (29 l/min) x (480 min/day) x (1/70 kg) = 86.5 mg/kg/day. This calculation assumes a human breathing rate of 29 l/min, human body weight of 70 kg, exposure for 8 hours and makes no adjustment for adsorption by inhalation.

Subchronic Exposure

Wolf et al. (1956) exposed guinea pigs, Rhesus monkeys, rabbits, and rats to concentrations of 400 ppm to 2,200 ppm ethylbenzene for 7-8 hrs/day, 5 days/wk, for up to 6 months. No effects were seen in guinea pigs, monkeys or rabbits. In rats, dose levels of 600 ppm and above caused increased liver and kidney weights and histopathological changes in the kidneys and liver, while 400 ppm produced only increased liver and kidney weights. ENVIRON has calculated a subchronic ADI for ethylbenzene based on the study by Wolf et al. (1956) in which 400 ppm was identified as the subchronic inhalation LOAEL in rats. The ADI was calculated as follows: (400 ppm) x (106.12/24,450) x (5 days/7 days) x (420 min/day) x (0.1 l/min) x (1/0.25 kg) x (1/200) = 1.04 mg/kg/day. A safety factor of 200 was used because mild adverse effects were seen at 400 ppm.

Chronic Exposure

The critical experiment for calculating a chronic ADI for ethylbenzene is a study of the effects of oral exposure in rats (Wolf et al. 1956). Rats received ethylbenzene in olive oil by gavage at dose levels of 13.6, 136, 408 or 680 mg/kg/day, 5 days/wk, for 6 months (182 days). Increases in liver and kidney weights as well as slight histopathological changes in these organs were observed at the two highest dose levels. No observable effects were noted in rats exposed to 13.6 or 136 mg/kg/day. Parameters examined included growth, mortality, appearance and behavior, hematology, terminal blood urea nitrogen (BUN) concentration, organ weights, body weight, bone marrow counts, and histopathology.

The USEPA (1986) has proposed an ADI/Rfd of 0.1 mg/kg/day based on the "subchronic to chronic" rat NOEL of 136 mg/kg/day and LOAEL of 408 mg/kg/day for the study at Wolf et al. (1956). A safety factor of 1,000 and a correction factor of 5/7 were applied (USEPA 1986). Similarly, ENVIRON has calculated a chronic ADI for the ethlybenzene based on the subchronic oral NOEL in rats of 136 mg/kg/day, as follows: (136 mg/kg/day) x (5 days/7 days) x (1/1000) = 0.097 mg/kg/day. The safety factor of 1,000 was applied for human extrapoltion from a subchronic animal study and the correction factor of 5/7 was applied to convert the five-day dosing regime to a seven-day exposure period.

H.11.3 Applicable and Relevant Standards

The recognized applicabel and relevant standards for ethylbenzene are summarized in Table H-11. The ambient water quality criterion for the protection of freshwater life is <32 mg/L. A proposed MCLG has been established at 0.08 mg/L. Regulations for workplace exposures have been developed by OSHA and ACGIH at 100 ppm or 435 mg/m^3. As previously noted, ethylbenzene is considered to be a noncarcinogen.

H.12 Chlorobenzene (ENVIRON, 1987)

H.12.1 Summary of Health Effects Data

Chlorobenzene (monochlorobenzene or MCB) is a central nervous system depressant. It may cause headaches, upper respiratory tract irritation, eye irritation, numbness, and eventual loss of consciousness in humans. In animals, sustained exposure to MCB can lead to liver and kidney damage. At higher levels, additional lesions can also be seen in bone marrow, spleen and thymus. At overtly toxic levels, shrinkage of the sperm-carrying ducts was demonstrable in dogs. MCB did not demonstrate carcinogenic activity at the levels tested in a National Toxicological Program (NTP) bioassay and has not been found to be mutagenic.

H.12.2 Toxic and Carcinogenic Effects

Acute Exposure

In humans, the only available information regarding the toxicity of chlorobenzene comes from individual case reports and occupational exposures to unknown concentrations. Reported effects have included signs of central nervous system depression, headaches, dyspepsia, and eye and respiratory irritation (Rosenbaum et al. 1947, Girard et al. 1969, Tarkhova 1965).

TABLE B-11

Summary of Toxicological Information for
Ethylbenzene

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (proposed)	none
EPA MCLG (proposed)	0.68
EPA Water Quality Criteria	
fish and drinking water	1.4
fish only	3.28
protection of aquatic life	<32
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA	100 ppm (435 mg/m^3)
ACGIH 8 hr TWA	100 ppm (435 mg/m^3)

Noncarcinogenic effects

risk characterization	
oral	
AIC	0.1 mg/kg/day
AIS	0.97 mg/kg/day
ADI	0.1 mg/kg/day
inhalation	
AIC	none
AIS	none
ADI	none
median effective dose	
oral	7.24×10^2 mg/day
inhalation	7.24×10^2 mg/day

Carcinogenic effects

Carcinogenic Potency Factor (10^{-6} risk)	
oral	none
inhalation	none
10% effective dose	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m^3	none
water (10^{-6} risk)	1.4
Classification, EPA	NC
Classification, IARC	3

In animals, acute inhalation exposures to high doses have resulted in respiratory tract irritation, narcosis, and central nervous system depression (DeCeaurriz et al. 1981). Acute exposures to high levels of chlorobenzene in animals have also produced adverse effects in the liver, porphyria, kidney toxicity, bronchiolar necrosis, and alterations in the flow of pancreatic secretions into the bile duct (Rimington and Ziegler 1963, Reid and Krishna 1973, Reid et al, 1973, Yang et al. 1979).

The TLV-TWA for chlorobenzene reported by ACGIH (1986) is 75 ppm (350 mg/m^3), although this limit is believed by some to be too high and is currently under review by ACGIH. The TLV can be converted to 69.6 mg/kg/day assuming a human body weight of 70 kg, breathing rate of 29 L/minute, and exposure for 8 hours per day.

Subchronic Exposure

Subchronic inhalation exposures in animals have produced effects such as reduced food consumption, liver kidney, and adrenal alterations, slight changes in some hematological parameters (Dilley 1977), decreased weight gain or weight loss, hypoactivity, conjunctivitis, aplastic bone marrow, seminiferous tuble atrophy (Monsanto 1978), and neurotoxicity (Tarkhova 1965).

Animals exposed to subchronic oral doses of chlorobenzene have developed weight loss; liver, kidney, gastrointestinal, and hematopoietic alterations (Monsanto 1978); increased porphyrin excretion; and neuropathy (NTP 1983).

The critical study used to develop a subchronic acceptable daily intake (ADI) is the rat inhalation study conducted by Dilley and coworkers (1977). Rats received exposures of 75 or 250 ppm MCB for 7 hours/day, 5 days/week for a total fo 120 exposures. Apparently, animals in both dose groups developed focal lesions of the adrenal cortex, kidney tubule lesions, congestion of liver and kidneys, and decreased SGOT. The LOEL identified from this study is 75 ppmm (0.34 mg/l). This can be converted to 40.8 mg/kg/day assuming a rat breathing rate of 0.1 L/minute, rat body weight of 0.25 kg, and expsure duration of 420 minutes/day for 5 days/week. A safety factor of 100 is employed for inter-and intraspecies conversion. An additional uncertainty factor of 10 is used to compensate for the use of a LOEL instead of a NOEL. The estimated subchronic ADI for inhalation exposure is 0.04 mg/kg/day.

Chronic Exposure

Chronic oral exposure to chlorobenzene has resulted in liver alterations including cytoplasmic basophilic changes and equivocal mild necrosis. Males exhibited a significant increae

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in neoplastic nodules in the high dose group (NTP 1983). The rats had received 0, 60, or 120 mg/kg by gavage, 5 days per week, for 103 weeks. Significantly decreased survival was reported for the high dose group. In the same study, mice received 0, 60, or 120 mg/kg (females) and 0, 30, or 60 mg/kg (males) 5 days per week. No treatment related effects were evident in the mice.

Reproductive effects have included gonadal effects in dogs including bilateral atrophy of seminiferous tubule epithelium, decreased spermatogenesis, and tubular atrophy (Monsanto 1978, 1967a). These effects occurred at relatively high doses, 2.0 mg/L vapor, 6 hrs/day, 5 days/week for 62 expsures i.e., 272.5 mg/kg/day for 13 weeks) which produced overt illness or death in the animals.

Unpublished results of a teratology inhalation study in rats and rabbits were devoid of significant teratological findings, even though high doses produced maternal toxicity (Hayes et al. 1982). Rats and rabbits were exposed to 0, 75, 210, or 590 ppm chlorobenzene for six hours per day during gestation.

A NOEL from the NTP (1983) rat study, of 60 mg/kg (5 days per week), is equivalent to 43 mg/kg/day. Using a safety factor of 100 for inter-and intraspecies conversion, a chronic ADI of 0.043 can be estimated. USEPA (1986) has reported an RfD/ADI of 0.03 for chlorobenzene. No basis was given, although there is some indication that the RfD/ADI is under reconsideration by USEPA.

H.12.3 Applicable and Relevant Standards

The recognized applicable and relevant standards for chlorobenzene summarized in Table H-12. The ambient water quality criterion for the protection of freshwater life is <0.25 mg/L. A proposed MCLG has been established at 0.006 mg/L. Regulations for workplace exposures have been developed by OSHA at 75 ppm or 350 mg/m^3. As previously noted, chlorobenzene is considered to be a noncarcinogen.

TABLE H-12

Summary of Toxicological Information for
Chlorobenzene

Relevant Requirements, Criteria, Advisories or Guidance	Value (mg/L)
EPA MCL (proposed)	none
EPA MCLG (proposed)	0.06
EPA Water Quality Criteria	
fish and drinking water	0.488
fish only	15.05
protection of aquatic life	<0.25
EPA Drinking Water Health Advisories	
1 day	none
10 days	none
chronic	none
OSHA 8 hr TWA	75 ppm (350 mg/m^3
ACGIH 8 hr TWA	75 ppm (350 mg/m^3)

Noncarcinogenic effects

risk characterization	
oral	
AIC	0.027 mg/kg/day
AIS	0.27 mg/kg/day
ADI	none
inhalation	
AIC	0.0057 mg/kg/day
AIS	0.053 mg/kg/day
ADI	none
median effective dose	
oral	5.6 x 10^1 mg/day
inhalation	7.18 x 10^1 mg/day

Carcinogenic effects

Carcinogenic Potency Factor (10^{-6} risk)	
oral	none
inhalation	none
10% effective dose	
oral	none
inhalation	none
Cancer Risk	
Inhalation at 1 ug/m^3	none
water (10^{-6} risk)	none
Classification, EPA	NC
Classification, IARC	3

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HAZARD ASSESSMENT OF INDICATOR CHEMICALS
IDENTIFIED AT THE TYSON'S DUMP SITE,
MONTGOMERY COUNTY, PENNSYLVANIA
by
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INTRODUCTION

The present endangerment assessment covers the off-site areas
surrounding the Tyson's Site in Montgomery County, Pennsylvania,
and relies to a large extent on procedures described by EPA for
analysis of public health risks at Superfund sites as outlined in
the draft Superfund Public Health Evaluation Manual. Basically,
this procedure consists of four steps: selection of indicator
chemicals, including the retrieval of basic toxicity data;
assessment of exposure concentrations, including estimation of
human intake; toxicity assessment; and risk characterization of
the indicator chemicals. These steps lead to the analysis and
development of design goals for proposed remedial alternatives.

The following evaluation concerns the identification and
evaluation of indicator compounds found off-site. Appropriate
selection and characterization of indicator chemicals for the
contaminants found at the Tyson's site is crucial to evaluating
alternatives for remedial action. Serious defects in
prioritization and/or in toxicological evaluation of the selected
chemicals will seriously affect the credibility of the final
conclusion.

Since the presence of proven or suspected carcinogens will have a
crucial impact on the selection of indicator chemicals, the
following discussion initially focuses on the assessment of
potentially carcinogenic compounds, and then evaluates relevant
non-carcinogens.

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I.   POTENTIAL CARCINOGENS

Two basic steps are involved in carcinogen risk assessment:

A.   Identification of carcinogens to be selected.
B.   Estimation of levels of risk for exposed populations.

The first step will deal mainly with the strength of evidence that a given compound is carcinogenic or not, and if so, to which animal species and under which circumstances.

A. Identification of Carcinogens to be Selected

The interpretation of carcinogenicity data from man and experimental animals involves issues of great complexity. Divergent opinions often exist within the scientific community on how to carry out a meaningful extrapolation.

A.1   Classification Systems

With the goal of providing governments and their advisors with authoritative scientific opinion on which to base preventive measures, The World Health Organization (WHO) International Agency for Research of Cancer (IARC) in Lyons in 1971 launched the program on the Evaluation of the Carcinogenic Risk of Chemicals to Humans - an effort actively supported by the U.S. government through financial support from the National Cancer Institute. This program involves the preparation and publication of monographs that evaluate individual chemicals as well as risks resulting from exposures to complex mixtures. The evaluations found in the IARC monograph for carcinogenic risk associated with human exposure to chemicals have found world-wide acceptance. EPA has modeled its system for stratifying the weight-of-evidence on the classification system developed by IARC (Federal Register 51(185) September 24, 1986, 33992-34003).

IARC makes a clear distinction between human carcinogens and experimental animal carcinogens. When human data are lacking for

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a substance, IARC makes no classification as to carcinogenicity
for humans. The National Toxicology Program (NTP), from which
EPA derives a   major part of its data base, also makes no
predictions as to the carcinogenicity of a chemical compound in
man based solely on the experimental data.

The European Common Market has recently implemented rules for the
assessment and classification of carcinogens in connection with
the labelling of chemical products which, like the EPA system,
have been based on the IARC weight-of-evidence concept. The
European rules are embodied in the 5th adaptation of the
so-called 6th Amendment of the European Communities' Council
Directive 67/548/EEC of July 29, 1983.

In spite of being based on a common philosophy and using
virtually the same data, the IARC and the competent authorities
within the European Communities have come to different
conclusions from the US EPA's as to the assessment of the
potential carcinogenicity of several compounds of concern here.
This difference of opinion derives mostly from different
interpretation of animal data, in particular of the relevance of
liver tumors in rodents. EPA's position on this issue has been
heavily criticized by the scientific community inside and outside
the US, and EPA policy seems to be undergoing a gradual change in
response to the criticism.

In a recent publication Elisabeth Anderson, then with EPA,
described this changing attitude in the following words:

> "...these so-called 'cancer-principles' [of EPA] received
> broad and general criticism by the scientific community, a
> substantial part of the private sector, and the Congress
> (e.g., see the Lancet, 1976). The major thrust of the
> criticism was not so much that these statements were
> incorrect, as it was that such a complex field as
> carcinogen assessment cannot be adequately covered in
> summary statements of this nature. More specifically, there
> was a widespread concern that the Agency would simply regard
> all agents associated with the induction of cancer in

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animals as equally likely to be potential human carcinogens;..."

For assessing evidence of carcinogenicity from experimental animal testing, IARC has developed the following classification system:

1. **Sufficient evidence of carcinogenicity** - indicates that there is an increased incidence of malignant tumors:

    a. in multiple species or strains, or

    b. in multiple experiments (preferably with different routes of administration or using different dose levels), or

    c. to an unusual degree with regard to incidence, site or type or tumor, or age at onset.

    Additional evidence may be provided by data concerning dose-response effects, as well as information on mutagenicity or chemical structure.

2. **Limited evidence of carcinogenicity** - means that the data suggest that the carcinogenic effects are limited because

    a. the studies involve a single species, strain, or experiment; or

    b. the experiments are restricted by inadequate dosage levels, inadequate duration of exposure to the agent, inadequate period of follow-up, poor survival, too few animals, or inadequate reporting; or

    c. the neoplasms produced often occur spontaneously or are difficult to classify as malignant by histological criteria alone, (for instance, lung and liver tumors in certain strains of mice).

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3.   <u>Inadequate evidence</u> - indicates that because of major
     qualitative or quantitative limitations, the data cannot be
     interpreted as showing either the presence or absence of a
     carcinogenic effect.

4.   <u>No evidence</u> - applies when several adequate studies are
     available which show that within the limits of the test used
     the chemical(s) is not carcinogenic.

It should be noted that the categories <u>sufficient evidence</u> and
<u>limited evidence</u> refer only to the strength of the experimental
evidence that these chemicals or complex mixtures are
carcinogenic and not to the extent of their carcinogenic activity
nor to the mechanism involved.

The degrees of evidence for carcinogenicity in <u>humans</u> are based
on epidemiological data and categorized by IARC as:

1.   <u>Sufficient evidence</u> of carcinogenicity indicates a causal
     relation between exposure and human cancer.

2.   <u>Limited evidence</u> of carcinogenicity indicates that a causal
     interpretation is credible, but that alternative
     explanations, such as chance bias or confounding, could not
     adequately be excluded.

3.   <u>Inadequate evidence</u> of carcinogenicity which applies to both
     positive and negative evidence, indicates that one of two
     conditions prevailed: a) there is little pertinent data; or
     b) the available studies, while showing evidence of
     association, do not exclude chance, bias or confounding.

4.   <u>No evidence</u> of carcinogenicity applies when several adequate
     studies are available which do not show evidence of
     carcinogenicity.

<u>At present, no objective criteria exist to interpret the animal
data directly in terms of human risk.</u> Thus, in the absence of
sufficient evidence from human studies, IARC makes an evaluation

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of the carcinogenic risk to humans based on consideration of experimental data.

The chemicals, groups of chemicals, or industrial processes are placed into one of three groups.

> Group 1 The chemical, group of chemicals, or industrial processes is/are carcinogenic for humans. This category is used only when there is sufficient evidence to support the causal association between the exposure and cancer.

> Group 2 The chemical or group of chemicals is probably carcinogenic for humans. This category includes chemicals for which the evidence of human carcinogenicity is "sufficient" as well as chemicals for which it is only suggested. To reflect this range, the category has been divided into higher (Group A) and lower (Group B) degrees of evidence. The data from experimental animal studies play an important role in assigning chemicals to Group 2, and particularly to those in Group 2B; thus, the combination of sufficient evidence in animals and inadequate data in humans usually results in a classification of 2B.

> Group 3 The chemical or group of chemicals cannot be classified as to its carcinogenicity for humans.

The EPA system is very similar to the IARC scheme, with Group A - substances carcinogenic to humans; and Group B - probable human carcinogens, featuring the same subdivisions B1 and B2. However, there are three groups corresponding to IARC's group 3: Group C - compounds possibly carcinogenic to humans; Group D - compounds which cannot be classified as to human carcinogenicity due to inadequate animal evidence of carcinogenicity; and Group E - no evidence of carcinogenicity for humans.

A.2   Supporting Evidence; Short-Term Tests

Positive results from short-term tests have been cited as supporting evidence for the possible carcinogenicity of compounds like trichloroethylene. However, the genotoxic activity is

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questionable or at best very weak and of little value when viewed
as supporting evidence for human carcinogenicity.

In December 1979 the OECD (Organization for Economic Cooperation
and Development)[1] Chemicals Group initiated the "Hazard
Assessment Project" to develop principles for performing,
a meaningful first assessment of the potential hazard of
chemicals based on the OECD Minimum Pre-marketing set of Data
(MPD).

Under the project, three Working Parties were established with
Lead country support, to work on Exposure Analysis (Germany),
Natural Environment Effects (Canada) and Health Effects
(United States).

It was felt that the establishment of internationally accepted
guides for interpretation of individual data elements, and in the
future for integrated assessments thereof, would significantly
reduce unnecessary divergences in hazard assessments from one
Member country to another. Serious impediments to trade in new
chemicals would thereby be avoided.

The Provisional Data Interpretation Guides, published in 1984 for
initial hazard assessment of chemicals, constitute a first

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[1]The OECD is a governmental organization, established by an
international convention in 1960. Its origins are in the
Organization for European Economic Cooperation, which was
established in 1948 to administer funds from the Marshall Plan.
Its members are 24 industrialized countries from North America,
Western Europe, Asia, and Oceania. The focus of the Organization
and its governing body is the Council, composed of permanent
representatives of the Member countries and chaired by the
Secretary-General. It is within the Council that governments
come together to approve, on a consensus basis, actions on
matters of substance. These actions usually take one of two
forms: Decisions, which are legally binding on Member countries,
and Recommendations, which, while not legally binding, carry a
strong moral obligation.

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important step towards mutual understanding of hazard assessments
and represent a provisional tool that can be used in Member
countries in their progressive implementation of the 1974 OECD
Council Recommendation and the 1982 Decision.

As to the role of short-term tests these guidelines take the
following position:

> "A correlation has been reported between the results of
> short term mutagenicity tests and the carcinogenic potential
> of certain classes of substances in mammals."..."The
> objectives of mutagenicity screening tests are to identify
> potential mammalian carcinogens, based upon the empirical
> correlation between certain short-term mutagenicity tests
> and carcinogenicity, and to identify potential mammalian
> mutagens."

According to the OECD expert group the only conclusion which can
be drawn from such studies is that positive results from
short-term tests "indicate that a chemical has the ability to
induce genetic damage and the potential of being a mammalian
carcinogen and/or mutagen." and that these results "may suggest
the need for further tests", (OECD, Data Interpretation Guides
for Initial Hazard Assessment of Chemicals, Paris, 1984,
pp. 58-60).

According to the IARC the evidence from short-term tests is
assessed and classified as follows:

1. "Sufficient evidence is provided by at least three positive
    entries, one of which must involve mammalian cells in vitro
    or in vivo and which must include at least two of three
    end-points - DNA damage, mutation and chromosomal effects.

2. Limited evidence is provided by at least two positive
    entries.

3. Inadequate evidence is available when there is only one
    positive entry or when there are too few data to permit an
    evaluation of an absence of genetic activity of when there

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are unexplained, inconsistent findings in different test systems.

4.   No evidence applies when there are only negative entries; these must include entries for at least two end-points and two levels of biological complexity, one of which must involve mammalian cells in vitro or in vivo."

IARC points out with regard to the interpretation of such results that,

1.   "At present, short-term tests should not be used by themselves to conclude whether or not an agent is carcinogenic, nor can they predict reliably the relative potencies of compounds as carcinogens in intact animals.

2.   Since the currently available tests do not detect all classes of agents that are active in the carcinogenic process (e.g., hormones), one must be cautious in utilizing these tests as the sole criterion for setting priorities in carcinogenesis research and in selecting compounds for animal bioassays.

3.   Negative results from short-term tests cannot be considered as evidence to rule out carcinogenicity, nor does lack of demonstrable genetic activity attribute an epigenetic or any other property to a substance."

A.3   Overview of Compounds Selected

The following suspected or proven carcinogens have been found in appreciable concentrations off the Tyson's site: aldrin, arsenic, benzo(a)anthracene, benzo(a)pyrene, bis(2-ethylhexyl)phthlate, DDT and trichloroethylene.

Of the compounds selected only arsenic is considered as proven human carcinogens and is classified as such by EPA (Group A) as well as by IARC (Group 1).

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Benzo(a)pyrene has been considered as a probable human carcinogen by IARC with a higher degree of evidence (Group 2A); EPA has rated the compound as (B2), whereas both IARC and EPA have classified DDT in the lower group (2B and B2, respectively).

For all the other substances, IARC has considered that insufficient evidence is available to make any kind of assessment as to carcinogenicity in humans (Group 3).

However, out of these chemicals IARC considers that there is sufficient evidence of carcinogenicity in experimental animals for benzo(a)anthracene (B2 according to EPA) and bis(2-ethylhexyl)phthalate (B2 according to EPA). For aldrin and trichloroethylene - considered as probable human carcinogens in Group B2 by EPA - presumption of carcinogenicity relies solely on very limited data derived from animal experiments, the relevance of some of which have been seriously questioned. The support has here been described by IARC as limited evidence of carcinogenicity to animals.

There are many reasons for the emergence of different opinions in the assessment of toxicological data, especially on issues which constitute a mixture of scientific opinions and administrative policy decisions. In my opinion, the divergences for some of the compounds treated here, notably the chlorinated hydrocarbons, seems mainly to derive from EPA's inappropriate utilization of available scientific data, in part violating general principles laid down by institutions on which EPA relies for scientific advice and information.

One special problem seems to be that EPA does not seem to consider mechanistic aspects when assessing carcinogenicity studies for the purpose of classification, but will strictly adhere to the rules laid down in the guidelines based on pathological evidence.

The qualitative evidence related to the potential carcinogens involved will be treated under four different sections:

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A.5 Chlorinated Hydrocarbons: Aldrin, DDT, and Trichloroethylene.

A.6 Polycyclic Aromatic Hydrocarbons: Benzo(a)anthracene, and Benzo(a)pyrene.

A.7 Peroxisome Proliferators: Bis(2-ethylhexyl)phthalate

A.4 Arsenic

The human carcinogenicity of these agents has been universally accepted.

Arsenic: Chronic exposure to inorganic arsenic in drinking water, drugs, and in occupational environments has been shown to be associated with the induction of skin cancer. Studies involving smelters and the production of arsenic-containing pesticides have provided strong evidence for an association between inhalation exposure to inorganic arsenic and lung cancer; and case reports have also suggested that arsenic may induce liver tumors (hemangioendothelial tumors or angiosarcoma) as well as blood dyscrasias.

Inorganic arsenic is unique in that the carcinogenic action has not been adequately reproduced in experimental animals.

A.5 Chlorinated Hydrocarbons: Aldrin, DDT, and Trichloroethylene

Three major objections can be raised concerning EPA's use of experimental data to classify the chlorinated hydrocarbons:

- Interpretation of tumor induction in rodents and its relevance to man.
- Choice of dose levels.
- Presence of genotoxic and/or carcinogenic impurities in test compounds.

A.5.1 Interpretation of Liver Tumor Induction in Rodents

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These compounds share the common toxicological feature of being
more or less potent hepatotoxins. The liver here constitutes a
main target organ for potential carcinogens as well as for
non-carcinogenic effects. In bioassays in rodents, liver cancer
has been induced in mice, but not in rats, by high doses of
aldrin, trichloroethylene and tetrachloroethylene. However, such
liver tumors occur spontaneously relatively often in mice, and it
is universally accepted that great caution should be exercised
when interpreting an increased incidence of such lesions in
treated animals. The main reason for this critical attitude is
that the increased incidence of liver tumors seen after high
doses of such compounds may be caused by a toxic insult to the
liver rather than due to a carcinogenic action of the chemical
per se.

The liver tumors of rodents, particularly of mice, are often
difficult to classify by morphological criteria leading to
considerable confusion in the literature. In one study 10 expert
pathologists made identical diagnoses in only 5 cases out of 43
possible (Essigmann, E.M., Mouse Hepatic Neoplasia. Diss.
Massachusetts Inst. Technology 1980).

Further, the spontaneous incidence of hepatic tumors may vary
widely between different strains and may reach 100% in males of
some strains. Also, different laboratories have reported large
variations in the spontaneous incidence of liver tumors within
the same strain. In one laboratory using the same strain these
incidences do not remain constant but show time-related trends.
Thus, for the B6C3F$_1$ mouse used by NTP - on which EPA has
depended for obtaining crucial qualitative as well as
quantitative data for carcinogen assessment - the historical
incidence of benign and malignant liver tumors was reported in
1979 to be 21% in males and 4% in females (Ward, J.M. et al, J.
Natl. Cancer Inst., 63(1979)849). In 1984 this rate had on the
average increased to 31% (range 18-47%) in males vs. 8% in
females (Haseman, J,K, et al, Toxicol. Pathol. 12(1984)126). Age
at autopsy is also critical. In one study this incidence tripled
in male mice between the 53rd and 78th week and from the 79th to
beyond the 104th week of age (Ward, J.M. et al, Natl. Cancer
Inst. 63(1979)849).

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Not surprisingly, a number of exogeneous and endogeneous factors like variations in diet, ingestion of promotors, hormonal imbalance, changes of the intestinal flora, or hepatectomy (surgical removal of part of the liver) will profoundly affect the spontaneous incidence of liver tumors in the mouse. Thus, in the classical experiments by Tannenbaum an increase of the fat content in the diet increased the liver tumor incidence from 36% to 51% in C3H mice (Tannenbaum, A., Cancer Res. 2(1942)468).

Finally, many experiments have demonstrated that this category of chlorinated hydrocarbons - similar to alcohol - exerts a promoting effect on rodent liver tumors induced by other factors.

In the monograph entitled "Long-Term and Short-Term Screening Assays for Carcinogens; A Critical Appraisal" IARC expands on this issue (WHO, IARC Monographs, Suppl.2, Lyon, 1980):

> "Some strains of animals have extremely high spontaneous tumor rates; for example, most Fisher F334 rats develop testicular tumors within two years, and some strains of mice likewise have very high spontaneous rates of liver, lung, and mammary tumors. Alterations in gross (Carroll & Khor, 1975) or even apparently minor (Roe & Tucker, 1973) aspects of diet can easily double or halve these spontaneous rates. It is thus uncertain how to interpret chemical carcinogenesis experiments in which, although there is a highly statistically significant effect of treatment, the chemical only affects the incidence of such tumors. In the absence of any further data, there is a general consensus that such a result does not provide sufficient evidence of carcinogenicity."

The degree of experimental variability encountered and the lack of uniform agreement by scientists and regulatory authorities concerning extrapolation of findings of liver tumors in mice led the well-known Nutrition Foundation - a non-profit institution dedicated to the advancement of nutritional science and its applications - to convene in 1982 an International Expert Advisory Committee on the Relevance of Mouse Liver as a Model for

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Assessing Carcinogenic Risks. This expert committee concluded under the heading "Regulatory Interpretation of Mouse Hepatoma":

"The usefulness of the mouse as a model for carcinogenicity testing for regulatory purposes continues to be a contentious issue. The concern over the mouse is directed, but by no means limited, to the propensity of certain commonly used strains to develop a high proportion of liver neoplasms with or without exposure to chemicals. Concern is heightened because of the large number of substances, representing a wide variety of chemical classes (Soderman, 1982), that induce these tumors. This high background incidence of liver tumors makes it difficult to determine whether a particular agent is actually inducing, or is merely enhancing, in a non-specific fashion, the incidence of tumors. It is now well established that some chemicals induce tumors only in mouse liver"... "Several strains of laboratory mice, including the B6C3F$_1$, have very high and variable spontaneous tumor incidences (Tarone et al. 1981). Irrespective of whether this is a result of genetic susceptibility or environmental factors, or both, it indicates that mouse liver contains a significant population of "initiated" or latent tumor cells. These cells would be expected to be susceptible to the promoting effects of cellular proliferation associated with chronic cytotoxicity. A similar susceptible population does not appear to exist in human livers in the Western hemisphere. Therefore the relevance to human populations at least in these regions of such enhancement of spontaneous tumor incidences in the mouse is questionable."... "From a regulatory point of view, caution is necessary in making judgments based upon tumor pathogenesis alone since we do not have a full understanding of carcinogenic mechanisms. For this reason, it is necessary that the total weight of evidence of carcinogenicity be considered in making regulatory decisions. There could be little doubt that it would be prudent to severely limit or even eliminate exposure to potently genotoxic chemicals which induce malignant tumors at multiple sites or in multiple species, and at low exposure levels. Less concern is warranted in the case of chemical

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induction of tumors only in mouse liver, particularly if the
tumors are primarily benign neoplasms, that are associated
only with high exposure levels that produce additional
biological effects such as chronic tissue injury."

In its monograph "Hepatocarcinogenises in laboratory rodent;
relevance for Man" The European Chemical Industry Ecology and
Ecotoxicology Center (ECEEC) strongly opposes the injudicious
extrapolation of findings of liver cancer in rodents caused by
hepatotoxic chemicals directly to man (ECETOC monograph No. 4,
Oct. 1982). The joint Food and Agriculture Organization - World
Health Organization (FAO-WHO) meeting on pesticides residues held
in Rome in 1984 concluded with respect to this issue,

> "It has been suggested that it would be unwise to classify a
> substance as a carcinogen solely on the basis of the
> species-specific increase of tumors which occurs
> spontaneously with high frequency. Moreover, when a
> chemical results in an increased incidence of liver tumors
> in mice, carcinogenicity observed in two species other than
> mouse would be appropriate."

Similar considerations also apply to the increase in lung tumors
in mice reported in two studies with trichloroethylene. Of
significance here are the concerns that the National Toxicology
Program (NTP) obviously has about the continued use of the $B6C3F_1$
mouse in carcinogen bioassays:

> "Because a substantial data base has been developed with the
> $B6C3F_1$ mouse, there is a natural reluctance to change the
> test strain to one that might present fewer problems. Yet,
> if the continued use of the present strain does not provide
> a level of discrimination to yield reasonably definitive
> results, its continued use must be weighed seriously.
> Important factors that should be considered in changing the
> test strain of mouse relate to evidence that the strain now
> used does not provide an adequate level of discrimination
> and that an alternative strain with superior characteristics
> in terms of longevity, responsiveness and level of
> spontaneous tumors has been identified." (Board of

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Scientific Counselors, National Toxicology Program: Report
of the NTP Ad Hoc Panel on Chemical Carcinogenesis Testing
and Evaluation, U.S. Dept. of Health and Human Services,
Aug. 17, 1984.)

Judging from EPA's response to public and science advisory board
comments on its recent cancer assessment guidelines, the Agency
seem to have given the massive criticism of its previous stand on
the issue of the mouse liver tumors some additional
consideration:

"A large number of commentors expressed opinions about the
assessment of bioassays in which the only increase in tumor
incidence was liver tumors in the mouse. Many felt that
mouse liver tumors were afforded too much credence,
especially given existing information that indicates that
they might arise by a different mechanism, e.g. tissue
damage followed by regeneration. Others felt that mouse
liver tumors were but one case of a high background
incidence of one particular type of tumor and that all such
tumors should be treated in the same fashion. The Agency
has reviewed these comments and the OSTP principle regarding
this issue. The OSTP report does not reach conclusions as
to the treatment of tumors with a high spontaneous
background rate, but states, as is now included in the text
of the guidelines, that these data require special
consideration. Although questions have been raised
regarding the validity of mouse liver tumors in general, the
Agency feels that mouse liver tumors cannot be ignored as an
indicator of carcinogenicity. Thus, the position in the
proposed guidelines has not been changed: an increased
incidence of only mouse liver tumors will be regarded as
"sufficient" evidence of carcinogenicity if all other
criteria, e.g., replication and malignancy, are met with the
understanding that this classification could be changed to
"limited" if warranted. The factors that may cause this
re-evaluation are indicated in the guidelines."

It should be underlined that, the EPA's position on the
evaluation of rodent liver tumors with high spontaneous incidence

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has not been consistent. In high doses selenium sulphides induce
an increase in hepatocellular carcinomas and adenomas in male and
female rats and female mice as well as pulmonary tumors in female
mice. NTP has judged the animal evidence of carcinogenicity as
sufficient. However, in this case the EPA has evidently taken
the position either that selenium may only pose a cancer risk at
high doses or that the rodent model is not valid here, and the
compound is not listed as a carcinogen in the Superfund Public
Health Evaluation Manual of December 18, 1985. Selenium is
consequently permitted as a feed additive in the US.

Finally, it should always be kept in mind that some of the
earlier NTP studies on which EPA has based its classifications
may need re-evaluation. Thus, the NTP board cited previously
does not seem to be quite satisfied with the quality of data
obtained in some of the sponsored studies;

"The introduction of GLP legislation has had a substantial
influence on the conduct and reporting of animal studies
carried out for regulatory purposes. Many previously
conducted bioassays and some ongoing NTP studies have not
yet benefited from detailed appraisals aimed at ensuring
their integrity. Since the credibility of regulatory
decisions on specific agents depends in large part on the
integrity and quality of the toxicology data base for that
agent, it is considered advisable that special attention be
focused on determining whether the interpretation of
previously conducted studies would be altered by the
application of GLP principles. The Panel recommends,
therefore, that the NTP staff consider auditing some of the
older studies that were pivotal to decision making and where
no new or additional studies have come forward in the
intervening years. The integrity of NTP test data and the
credibility of the NTP program are of fundamental importance
not only to the regulatory process but also to the science
of toxicology. With this in mind, the Panel also recommends
that the NTP staff consider other approaches for ensuring
the integrity of test data and maintaining the credibility
of all such programs. Such approaches might include, for
example, the testing of an agent known to be a human

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carcinogen but not previously tested in animals and the validation of a previously tested animal carcinogen by retesting."

A.5.2 Choice of Dose Levels

This brings me to the second major objection to the way experimental cancer data have been used by EPA in this context. When reviewing the cancer studies of trichloroethylene, many scientists in academia and industry have pointed out that tumors can have only been produced in experimental animals by exposure to these compounds in massive doses. The exposures that concern us here, for instance, are several orders of magnitude lower. Further, the high dose levels used to elicit toxic responses in the animals sometimes surpass the maximum tolerated dose (MTD). In its recent review of chemical carcinogens, the Office of Science and Technology Policy (OSTP) of the Executive Office of the President points out the following with respect to this question (FR March 14, 1985, p. 53),

"The concept and operational definition of the MTD have undergone considerable modifications during the last several years. The 1971 FDA Advisory Committee on protocols recommended that testing should be done at doses and under experimental conditions likely to yield maximum tumor incidence".

The OSTP points out that

"These recommendations have been controversial because high doses may themselves produce altered physiological conditions which can qualitatively affect the induction of malignant tumors. Normal physiologic homeostasis and detoxification, or repair, mechanisms may be overwhelmed and cancer which otherwise may not have occurred is induced or promoted."

OSTP continues,

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"Both the National Cancer Advisory Board Subcommittee
(NCAB) and the Interagency Regulatory Liason Group (IRLG)
cautioned against use of a dose so high that it produces
"unwanted toxic side effects" (IRLG) or "unphysiological
conditions which may in themselves enhance tumor formation
(NCAB)".

This viewpoint is also reflected to some extent in the recently
published EPA guidelines for carcinogenicity studies:

"Positive studies at levels above the MTD should be
carefully reviewed to ensure that the responses are not due
to factors which do not operate at exposure levels below the
MTD. Evidence indicating that high exposures alter tumor
responses by indirect mechanisms that may be unrelated to
effects at lower exposures should be dealt with on an
individual basis. As noted by the OSTP (1985), "Normal
metabolic activation of carcinogens may possibly also be
altered and carcinogenic potential reduced as a consequence
(of high-dose testing)." (FR 51, No. 185, Sept. 24, 1986,
p. 33995).

The toxic effects (including a mitogenic action) on the liver
provoked by the high doses of trichloroethylene employed in the
cancer studies certainly meet the criterion of an unphysiological
condition which may in itself enhance tumor formation.

A.5.3 Presence of Impurities in the Test Compound

The recent EPA guidelines for the testing of carcinogens are to a
large extent based on the reviews made by the Office of Science
and Technology Policy, Executive Office of the President. In its
latest review of February 1985 it is explicitly stated under the
heading "False Positives" with respect to the purity of the
compound to be used in the bioassays: "Appropriate precautions
in the design and evaluation of bioassays can minimize the chance
of false positive results. The test substances must be free of
carcinogenic contaminants and appropriate doses and routes of
exposure must be used [emphasis added here]" (OSTP, Chemical
Carcinogens; A Review of the Science and its Associated

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Principles, Feb. 1985, p. 56). The presence of the potent genotoxic agents, 1,2-epoxybutane and epichlorohydrin, as stabilizers in several studies covering the compounds discussed here does not increase confidence in the positive results obtained.

A.5.4   Aldrin

Upon oral administration, aldrin has induced malignant liver tumors in mice and thyroid tumors in rats. The animal data available has been judged as limited, and evidence from short-term tests as inadequate by IARC. The objections raised against EPA's classification of aldrin as a probable group 2B carcinogen are similar to those raised with respect to the halogenated hydrocarbons discussed below, and the inadequacy of the mouse liver tumor model will, thus, be discussed in detail under the section A.5.6 - Trichloroethylene.

As to the thyroid tumors in the rat, these tumors may readily be explained in terms of a promotive effect where the chlorinated hydrocarbon interferes with the normal thyroid hormone status which indirectly causes compensatory thyroid hyperplasia eventually giving rise to neoplasia. Mechanistically, thyroid tumor induction by aldrin seems to be related to the effects of an iodine-deficient diet in the rat, which is a potent inducer of such tumors and is carcinogenic by itself (cf. Oshima, M. and Ward, J.M.; Dietary Iodine Deficiency as a Tumor Promoter and Carcinogen in Male F344/NCR Rats, Cancer Res. 46(1986)877-883). Consequently, John Weisburger has classified aldrin as an epigenetic carcinogen of the promoter type (cf. Casarett and Doull's Toxicology, 3rd Ed. MacMillan 1986, p. 124).

A.5.5   DDT

Upon oral administration DDT has been found to be carcinogenic in mice, inducing benign and malignant liver neoplasms, lymphomas and lung neoplasms. Hepatomas have also been found to occur in rats after administration via this route. Two feeding studies with hamsters, and corresponding investigations in dogs and monkeys have given negative results. DDT does not seem to

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possess genotoxic properties, and evidence from short-term test have been judged as inadequate by IARC. The types of tumors found in the mouse often occur spontaneously and DDT has also been shown to possess promoting activity. Some authorities therefore consider DDT as an epigenetic carcinogen of the promoter type (cf. Casarett and Doull's Toxicology, 3rd Ed. MacMillan 1986, p. 124). On the other hand liver neoplasms have been unequivocally induced in two animal species, and both IARC and EPA have placed DDT in the category of probable human carcinogens (although in the lower group with respect to the power of supporting evidence). At the 1984 Joint Meeting of the FAO Panel of Experts on Pesticide Residues in Food and the Environment and the WHO Expert Group on Pesticide Residues, the tumors induced by DDT in rodents were not considered relevant to man, but were ascribed to differences in metabolism and to the peculiar sensitivity of rodent liver. Great importance was attached to the fact that long term studies in monkeys (up to 11 years) had failed to provide any evidence of carcinogenicity, as well as the fact that no conclusive evidence for the human carcinogenicity of DDT has yet emerged in spite of the widespread use of DDT causing high exposures to large population groups over a long period of time. An ADI of 0.02 mg/kg/day has been established for DDT by FAO/WHO.

A.5.6   Trichloroethylene

The problems involved in interpreting increased incidences of liver tumor in rodents have already been discussed. In its conclusions to the EPA health assessment document for trichloroethylene EPA reaffirms its questionable position with regard to this issue (EPA-600/8-82-006F, July 8, 1985):

> "The U.S. Environmental Protection Agency's proposed guidelines for carcinogen assessment (US EPA 1984) regard the mouse-liver-tumors-only-response as sufficient evidence for carcinogenicity in animals." and "Based on EPA's proposed cancer guidelines, the overall evidence for TCE results in the classification of B2, that is a probable human carcinogen."

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As to the mutagenicity of TCE, EPA expresses the opinion that data on pure TCE do not allow any conclusion, but that in case the compound is at all mutagenic, it must be a very weak mutagen.

Without mentioning 1) that IARC actually had in fact evaluated the results from relevant studies as only "limited evidence of carcinogenicity in mice" and disregarding 2) the clear position which IARC has taken on the inadequacy of evidence based solely on liver tumors in this species, and further disregarding 3) the IARC's well-publicized policy of not to make any statement as to the carcinogenicity to man when data from humans are lacking, EPA makes the following curious and misleading statement:

> "the TCE carcinogenicity results could be classified under the criteria of the International Agency for Research on Cancer as either: "sufficient" or "limited" depending on which of the different current scientific views about chlorinated organic compound induction of liver tumors in mice is chosen. Since there are no adequate epidemiological data on humans, the overall ranking of TCE under the criteria of IARC depends primarily on the position taken regarding the mouse liver tumor. Thus, the overall ranking could be either Group 2B or Group 3. The more conservative Public Health view would regard TCE as a probable human carcinogen (Group 2B), but there is also scientific sentiment for regarding TCE as an agent that cannot be classified as to its carcinogenicity to humans (Group 3)."

When metabolized in the liver, trichloroethylene is converted to trichloroacetic acid. Trichloroacetic acid induces peroxisome proliferation and a mitogenic response in the rodent liver that is known to be associated with induction of hepatocellular carcinoma in the rodent, a process which occurs by a non-genetic so-called "epigenetic mechanism".

In its interpretation of experimental data EPA avoids the issue of the significance of the metabolic formation of trichloroacetic acid which in turn induces proliferation of the peroxisomes in the rodent liver. In his address to the 1986 British Industry Biological Research Association (BIBRA) Annual Scientific Meeting

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held on 14 October 1986 at the famous Royal College of
Physicians, Dr. Purchase of the Central Toxicology Laboratory at
Imperial Chemical Industries discussed the position of the United
States EPA on trichloroethylene. He pointed out that
trichloroethylene is metabolized predominantly to trichloroacetic
acid which appears to be the active metobolite in both rats and
mice and that the administration of trichloroethylene to mice
results in a substantial increase in the number of peroxisomes
present in the hepatocytes, whereas human hepatocytes do not
produce any increase in peroxisomes after administration of
trichloroacetic acid. He also said that there are a number of
chemicals that induce liver cancer in rodents after inducing
peroxisome proliferation, suggesting that the induction of
peroxisomes is a necessary step for the induction of
hepatocellular carcinoma after administration of these chemicals.
Dr. Purchase concluded,

> "This evidence on the mechanism by which trichloroethylene
> produces cancer in mice and the fact the mechanisms do not
> operate in human cells suggest that it is unlikely that
> trichloroethylene will be carcinogenic in man. The general
> conclusion is supported by the limited epidemiological
> evidence that is available. In spite of this information,
> trichloroethylene is now controlled as a carcinogen in the
> U.S.A. using risk assessment techniques that assume a
> non-threshold dose-response and human sensitivity equal to
> that of the mouse". (Food and Chem. Toxicology, Vol. 24,
> No. 4, 1986, pages 345-347)

Finally, it should be mentioned that a number of epidemiological
surveys have been conducted to examine potential carcinogenicity
of this compound in occupationally exposed populations, involving
thousands of workers. In spite of heavy exposure to this
population, no significant increase in the incidence of liver
cancer or of any other type of neoplasia has been found. The
slight excess of genital/urinary cancers and lymphomas found in a
few studies are difficult to interpret but may have been caused
by confounding factors.

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According to the IARC, the evidence available from short-term tests is to be regarded as inadequate.

A.6 Polycyclic Aromatic Hydrocarbons (PAHs)

Because of many common features the polycyclic hydrocarbons, PAHs, are conveniently treated together. Several representatives of the class are stongly implicated in the proven association between smoking and lung cancer, between occupational exposure to coke-oven emissions, coal-tar, pitch, mineral oils and similar products and cancer of skin, lung, bladder and the gastro-intestinal tract. However, because exposure involves complex mixtures where, clearly, there is interaction between a number of initiators and promoters, it ismore difficult to assess the activity of individual members of the group. Here, the relative potency exhibited in animal experiments - mainly skin application (alternatively subcutaneous or intramuscular injections) in mice - have been used as the only practical tool for the assessment of carcinogenicity. It should be pointed out, however, that the more active congeners do induce tumors at multiple sites. Several members of the group have shown to be genotoxic in a number of systems requiring metabolic activation.

Since the carcinogenic activity of PAHs are linked to the metabolic formation of reactive intermediates (e.g. dihydrodiol epoxides) of certain pathways, where the end effect depends on a delicate balance between activating and inactivating reactions, carcinogenicity is strongly dependent on species related metabolic differences. Many rodent species, like the mouse, are extremely sensitive to chemical carcinogens of this type, and experience from epidemiological investigations tend to demonstrate a much lower sensitivity of e.g. human skin than that of the mouse, a finding which has been corroborated by investigations in monkeys.

Benzo(a)anthracene. Whereas anthracene inself lacks carcinogenic activity, benzo(a)anthracene seems to possess such activity in so for as the compound has been shown to constitute a complete carcinogen for the mouse skin and has also shown activity by other routes of administration. Consequently, IARC has

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classified benzo(a)anthracene as a probable human carcinogen in Group 2B.

Benzo(a)pyrene constitutes one of the most extensively studied member of the PAH group and has been shown to be a local as well as a systemic carcinogen by several routes of administration. However, some comments seem to be pertinent when assessing the relevance of the more extensive bioassays involving oral administration (Neal, J. and Rigdon, R.H., Texas Rep. Biol. Med. 25(1967)553-557; Federenko, Z.P. and Yanisheva, N.Y., Gig. Sanit. 31(1966)168-173). The tumors found in these studies were localized in the forestomach of the mouse which – in contrast to the corresponding fundus portion of the human stomach – is nonglandular and more similar to the epithelium of the skin. Thus, the relevance of these studies are questionable as to this particular site. Other evidence, however, leave little doubt that this subtance should be classified as a probable human carcinogen as done by EPA and IARC.

Benzo(a)pyrene has often been used as indicator substance for the collective group of PAHs for the purpose of air quality monitoring, etc. The suitability of this choice may be questioned in view of the chemical instability of the substance, resulting in large variations in the relation between the concentration of this substance and the total amount of PAHs present.

A.7   Peroxisome Proliferators; Bis-(2-ethylhexyl)phthalate

In recent NTP bioassay bis-(2-ethylhexyl)phthalate (DEHP) was demonstrated to induce liver tumors in Fischer 344 rats and in B6C3F1 mice at very high doses. DEHP represents a distinct class of chemically unrelated carcinogens that share the property of inducing peroxisome (microbodies) proliferation in the mammalian liver (Reddy, J.K. and Lalwani, N.D. CRC Critical Rev. Toxicol. 12(1983)1-58). The only distinctive common chemical feature seems to be that all members of this class are carboxylic acids, or can be metabolically converted into a carboxylic acid. Extensive testing has demonstrated that DEHP completely lacks genotoxic activity, and it has been proposed that the

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carcinogenic action occurs by means of indirect processes linked
to the proliferation of the peroxisomes.

In the induced cell organelles, the beta-oxidation exzymes
responsible for degradation of fatty acids with the simultaneous
generation of hydrogen peroxide, are induced to a much greater
extent than the enzymes systems - mainly catalase - which utilize
peroxide as a substrate. There is also evidence of interference
with the function of intracellular protective thiols. Such an
imbalance is not evident in peroxisome proliferation induced by
e.g. hypothermia. It has therefore been suggested, that the
increased generation of peroxide - or free radicals derived
thereof - may cause DNA damage which together with the mitogenic
action of these compounds may induce neoplasia in the rodent.
Since peroxisome proliferation represents a, pathological effect
with a definite dose-threshold, it is generally accepted that
DEHP and similar substances cannot be assessed in the same way as
most other carcinogens.

DEHP, and some other peroxisome proliferators, probably
represents the best documented examples of epigenetic carcinogens
so far discovered. No evidence of carcinogenic effects in humans
is available and IARC has classified DEHP in Group 3. EPA has
placed DEHP under B2.

A.7 Concluding Remarks on Choice of Indicator Carcinogens

In summary, the general classification of aldrin,
bis-(2-ethylhexyl)phthalate, and trichloroethylene as probable
human carcinogens appears to lack adequate scientific support and
seems to be in conflict with assessments made elsewhere, for
instance by the IARC and by the competent bodies within the
European Common Market. The characterization of DDT as a B2
compound is also disputed. The lack of carcinogenic activity of
the chlorinated hydrocarbons at non-toxic levels is supported by
a recently published unique carcinogenicity study in rats with a
mixture of eleven volatile halogenated hydrocarbons in drinking
water carried out by the well-known National Institute of Public
Health and Environmental Hygiene in Bilthoven, The Netherlands.
In this study which in the author's opinion seems much more

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relevant to the present discussion than some of the NTP studies
cited above a mixture of the following substances was
administered to Wistar rats during a lifetime: trichloromethane,
tetrachloromethane, monobromodichloromethane, trichloroethylene,
tetrachloroethylene, 1,2-dichlorobenzene, 1,3-dichlorobenzene,
1,4-dichlorobenzene, 1,2,3-trichlorobenzene,
1,2,4-trichlorobenzene, and 1,3,5-trichlorobenzene. The total
concentration of the chlorinated hydrocarbons present in the
drinking water was high (up to a total of 22 mg/l), but not so
high as to elicit evident toxic effects. Under these conditions
no significant increase in the incidence of tumors could be
detected.

Naturally this does not exclude the possibility that these
compounds may have a tumor-promoting action in man similar to
that in the mouse in doses which elicit pathological changes of
the liver. However, the EPA's extrapolation of data, based on
this questionable effect in mice exposed to massive toxic doses
in order to calculate effects at low exposures where liver injury
is not observed, cannot be regarded as relevant.

Responsible scientific agencies of international repute, like
IARC, will not pass judgment on the carcinogenicity of substances
for which adequate data are not available and until information
has been produced which provides adequate evidence of
carcinogenicity, this substance should be treated as a
non-carcinogen.

In conclusion, of the selected compounds, only arsenic and the
polycyclic aromatics, benzo(a)anthracene and benzo(a)pyrene,
would appear to serve us as an appropriate indicator chemicals in
a quantitative risk assessment for carcinogenicity in this
context.

B. Estimation of Levels of Risk to Exposed Populations

Although a chemical or chemical mixtures has been identified as a
human carcinogen, this does not necessarily mean that the risk to
the individual in handling such a chemical or mixture is high.
The extreme differences in potency which exist between various

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carcinogenic agents must be emphasized. Also the high levels of
carcinogens normally present in food which evidently account for
a large part of the normal cancer incidence should also be
recalled in this context. When discussing lifetime risks in the
one part per million range, it should be emphasized that the
normal lifetime risk for cancer in absence of any significant
exposure to "industrial chemicals" lies in the order one in four
to one in five. In assessing cancer risks the quantitative
estimation of the level of risk is evidently crucial. Thus the
former member of the Board of Directors of the National Cancer
Institute and, a member of the National Academy of Sciences,
Professor Bruce Ames, said in his testimony to the Senate
Committee on Toxics and Public Safety Management on November 11,
1985 "every meal is full of carcinogens and when one compares the
level of carcinogens in contaminated water (or pesticides
residues in food) to the level of natural carcinogens, it is
clear that water pollution represents a trivial risk".

B.1 Choice of Mathematical Extrapolation Model

As was pointed out by OSTP:
> "No single mathematical procedure is recognized as the most
> appropriate for low-dose extrapolation in carcinogensis.
> When relevant biological evidence on mechanism of action
> exists (e.g., pharmacokinetics, target organ dose), the
> models or procedures employed should be consistent with the
> evidence."(Office of Science and Technology Policy, FR.
> March 14, 1985, Part II p. 9).

Among the numerous models used for extrapolation from high dose
to low dose, EPA recommends the use of a linearized multistage
model "in absence of information to the contrary" (FR, No. 185,
Sept. 24, 1986, pp. 33992-34003). The National Toxicology
Program ad hoc Panel on Chemical Carcinogenesis defined one of
the major problems in its report of August 17, 1984 in the
following way (p. 175):

> "Precise elaboration of the shape of the dose-response curve
> is generally not possible with an experiment of the
> magnitude of the current NTP bioassay."

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In the EPA document, however, it is said that "Different extropolation models guidelines may fit the observed data reasonbly well but may lead to large differences in the project risk at low doses."

EPA acknowledges that a risk estimate based on the linearized multi-stage model "does not necessarily give a realistic prediction of the risk. The true value of the risk is unknown, and may be as low as zero." Further, it is emphasized that

> "When pharmacokinetic or metabolism data are available, or when other substantial evidence on the mechanistic aspects of the carcinogenesis process exists, a low-dose extrapolation model other than the linearized multistage procedure might be considered more appropriate on biological grounds."

Adequate support for EPA's linearized multistage and closely related models has only been derived from experiments with potent genotoxic carcinogens, for instance, the induction of liver tumors (not kidney tumors) by 2-acetylaminofluorine. For such compounds the choice of this model would seem justified. However, this does not apply to the chlorinated aliphatic hydrocarbons like trichloroethylene, tetrachloroethylene, carbon tetrachloride or chloroform, where a hepatotoxic promoting action at high doses causes an increase in spontaneously occurring neoplasms. Therefore, the cancer potency factors claimed for these compounds in all probability represent gross over-estimations of risk, resulting in values in the same range as benzene - a proven human carcinogen. If indeed trichloroethylene constitutes a human carcinogen of similar potency, one would have expected a drastic increase in cancer incidence in several of the epidemological studies already performed, e.g. in some studies carried out in Sweden involving large cohorts of workers exposed to trichloroethylene at levels sometimes exceeding one hundred parts per million.

In summary, if halogenated hydrocarbons like the one discussed here should at all be considered as carcinogens, the carcinogenic

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potency factors are probably lower than those quoted by at least
an order of magnitude.

The proposed EPA guidelines were recently criticized by the
Office of Management and Budget (OMB). W.L. Gramm, head of OMB's
Office of Regulation and Regulatory Affairs, has stated in a
letter to EPA:

> "If worst-case assumptions and upper-bound estimates are
> used and repeatedly combined, they will produce a final risk
> estimate that is almost certain to amplify the real risk
> hundreds or thousands of times."

According to Gramm's letter other risk models should also be used.
He points out that the recommended model not only gives high risk
estimates when the data indicate high risks, but also is applied
in cases where data are questionable. Evidently EPA agrees with
OMB in principle but has emphasized that these concerns have
already been considered during the review of the proposed
guidelines (C&EN Sept. 15, 1986, p. 18-19). In its response to
public and science advisory board comments, EPA underlined that

> "The major criticism was the perception that EPA would use
> only one method for the extrapolation of carcinogenic risk
> and would, therefore, obtain one estimate of risk. Even
> commentors who concur with the procedure usually followed by
> EPA felt that some indication of the uncertainty of the risk
> estimate should be included with the risk estimate.
>
> The Agency feels that the proposed guidelines were not
> intended to suggest that EPA would perform quantitative risk
> estimates in a rote or mechanical fashion. As indicated by
> the OSTP report and paraphrased in the in the proposed
> guidelines, no single mathematical procedure has been
> determined to be the most appropriate method for risk
> extrapolation."

However, in practice the kind of graded approach advocated by OMB
and by others using alternative risk models never seems to be
used by EPA, in spite of the fact that available data would

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indicate other extrapolation procedures. Personally, the author
is not aware of any single carcinogen where EPA has used a less
conservative option than the linearized multistage model.

B.2.1 Arsenic

In the Superfund Public Health Evaluation Manual of December 18,
1985 a potency factor of 15 (mg/kg/day)-1 is given for exposure
by the oral route (lifetime unit risk due to 1 ug As/L in the
drinking water = 4.3 x 10-4) and 50 (mg/kg/day)-1 for inhalation
(lifetime unit risk due to continuous exposure to 1 ug As/m³ =
1.25 x 10-3 to 7.6 x 10-3). The risk estimates for inhalation
were based on studies on lung cancer in 2 populations of smelter
workers in the US, whereas the cancer risk from exposure via the
oral route was based on studies in a population in Taiwan known
to consume arsenic containing drinking water. The imprecision of
EPA's risk estimates is underlined in the Health Assessment
Document for Inorganic Arsenic (EPA-600/8-83-021F): "While it is
unlikely that the true risks would be higher than these
estimates, they could be substantially lower." One important
source of error in the lung cancer studies is the synergistic
effect between smoking and exposure to arsenic.

Inorganic arsenic compounds are unique in so far as they do not
appear to induce mutations but cause induction of chromosomal
aberrations in Drosophila and mammalian cells as well as SCE.
IARC has considered that limited evidence for activity in
short-term tests is available. The mechanism of action is
unknown, but evidence has been obtained indicating that arsenic
may interfere with error-prone DNA repair (Jung, E. et al, Ger.
Med. Mon. 14(1969)614-616; Jung, E., Haupt. Geschlechtskr.
46(1971)35-36; Rossman, T.G. et al, Env. Hlth. Perspect.
19(1977)229-233); Rossman, T.G., Mut. Res. 91(1981)207-211) as
well as causing immunosuppression (National Academy of Sciences,
Arsenic, NAS, Washington D.C., 1977). Arsenic may, therefore,
act as a co-carcinogen rather than as an initiator. The
promotive effect of arsenic trioxide on the induction of lung
tumors by benzo(a)pyrene in hamsters and rats as well as the
potentiating effect of arsenic on smoking-induced lung cancer
(Cancer, Report from the Swedish Cancer Committee, SOU 1984:67,

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Chapter 4.6, Stockholm 1984) seem to support such a conclusion.
Thus, the use of the linearized multistage model for calculation
of cancer risk for arsenic seems highly questionable. However,
since arsenic is an established human carcinogen it would seem
prudent to use the EPA estimates for upper limits of risk.

B.2.3 Polycyclic Aromatic Hydrocarbons

In this context it should perhaps be remembered that there is a
considerable normal intake (EPA cites 6 ug/day as an upper limit
for certain populations) of PAHs, e.g. by consumption of grilled
meat, etc. In addition, the quantification of risk from
exposures to mixtures of PAHs is extremely difficult. Based on
animal experiments with benzo(a)pyrene, potency factors have been
obtained. Risk estimates have also been derived from
epidemiological studies utilizing this substance as an indicator.
In the Superfund Public Health Evaluation Manual no quantitative
data are provided for risk assessment of benzo(a)anthracene. For
benzo(a)pyrene a potency factor of 11.5 (mg/kg/day)-1 is given
for the oral route, the corresponding value for inhalation being
set at 6.1 (mg/kg/day)-1. For quantification of cancer risk from
PAHs the Swedish Cancer Committee in part based its risk
assessment on data presented by Pike et al, (Pike, M.C. et al in
Fraumeni, J., ed. Persons at High Risk of Cancer, Academic Press,
New York, 1981, p. 317). The value utilized by the Committee for
lifetime risk upon continuous inhalation of 1 ug of
benzo(a)pyrene/m^3 (as indicator substance of all PAHs formed upon
combustion) was 0.7.

If the mixture of PAHs found at the Tyson site approximately
corresponds to what is typically found in products of combustion
processes, a similar approach may be considered.

II. NONCARCINOGENS

The following noncarcinogens have been identified in significant
quantities at the Tyson dump site: Carbon disulfide,
Di-n-butylphthalate, 1,1-Dichloroethane,
cis-1,3-Dichloropropene, Monochlorobenzene,
1,2,4-Trichlorobenzene, Phenol, Selenium, and Zinc.

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The paragraphs below give a brief presentation of the toxicology
of these agents and of the standards applicable to risk
characterization of potential exposures from the site.

A. Carbon Disulfide

The toxicology of carbon disulfide is well-known and includes
local as well as systemic effects involving several target organs.
In higher concentrations the vapors cause severe irritation of
eyes and respiratory tract and direct contact with the liquid has
a corrosive action on skin. Further, skin absorption of carbon
disulphide may be considerable, causing systemic effects.

Upon absorption very little carbon disulfide is excreted
unchanged. Metabolic conversion involves conjugation with
glutathione and certain amino acids, as well as oxidation and
excretion of various sulphur-containing compounds with the urine.
Of the conjugation reactions, reaction with glycine to give
dithiocarbamate and dehydration of the latter to yield
2-thio-5-thiazolidinone have attracted attention because of the
chelating properties of these matabolites. It has thus been
proposed that the neurotoxicity of carbon disulfide could be
explained in terms of chelation of metals essential for the
activity of enzymes such as dopamine hydroxylase (McKenna, M.J.
and DiStefano, V.J. Pharmacol. Exptl. Therap. 202(1977)253-266).
Other potentially toxic metabolites are isothiocyanates.

The main target organs for intoxication by carbon disulfide are
the central and peripheral nervous systems, the eyes and the
cardiovascular system. The action of carbon disulfide on the
nervous system involves toxic effects resembling "type 1 anoxic
damage" similar to those induced by e.g. azide as well as a
neurotoxic action targeted to the peripheral motor nerves giving
rise to axonal degeneration. Chronic exposure to high levels of
carbon disulphide induces a number of subjective symptoms like
irritability, memory impairments, sleep disturbances and loss of
motor coordination, and may result in a severe encephalopathia
characterized by psychotic symptoms (delirium, seizures and
mental impairment). In young persons a "Parkinsonian-like

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syndrome" has been described.   Pathological postmortem findings
have included neuronal degeneration and cell loss in the cerebral
cortex and the corpora striata.

At lower levels of chronic exposure to carbon disulfide,
polyneuritis may be induced with lower-extremity weakness and
paraesthesias, a condition which resembles thiamine deficiency.
Axons seem to be affected to a greater extent than the myelin
sheaths.   Hearing loss to high-frequency tones represents a
rather common sequelae to carbon disulfide intoxication.

From Finnish epidemiological studies (Tolonen, M. et al, Scand.
J. Work Environm. Hlth. 5(1979)109-114) it seems that chronic
exposure to carbon disulfide may contribute to coronary heart
disease (atherosclerosis of the blood vessels of brain,
myocardium, and glomeruli). These observations are supported by
the finding that carbon disulfide greatly accelerates the
formation of atheromas in rabbits that are given high cholesterol
diets.   The mechanism of acceleration of atherosclerosis has been
thought to involve direct injury of the epithelium of the vessels
coupled with metabolic changes induced by thiocarbamate
metabolites which have antithyroid effects (Van Stee, E.W. in
Cardiovascular Toxicology (Van Stee, E.W. Ed.), Raven Press N.Y.
1982, pp.1-35).

Effects on the eye, involving disturbances in the blood
circulation of the ocular fundus and the retina with central
scotoma and drop in visual acuity illustrates another toxic
potentiality of carbon disulfide. Subtle effects on the eye
involving changes in the microcirculation have been described at
exposure levels presumed to be safe (Raitta, C. and Tolonen, M.
in Neurotoxicity of the Visual System (Merrigan, W.H. and
Weiss, B. Eds), Raven Press, N.Y. 1980).

Other reported specific effects include chronic gastritis,
impairment of endocrine activity (adrenal, testicular) which may
result in toxic effects on male and female reproduction, abnormal
erythrocytic development (hypochromic anaemia), as well as
possible liver dysfunction.

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Risk Assessment: The Draft Superfund Public Health Evaluation
Manual of December 18, 1985 gives a minimum effective dose (MED)
for selection of indicator chemicals of 33 mg/kg and an AIC value
of 0.1 mg/kg/day for risk characterization of this chemical.

The NIOSH-recommended TWA is 1 ppm (3 mg/m^3) with a STEL of 10
ppm, whereas the corresponding ACHGIH TWA is 10 ppm. According
to NIOSH, workers should also be advised of the potential effects
on the reproductive system. Assuming that the NIOSH TWA value
represents the highest no-observed-adverse-effect-level (NOAEL),
and assuming a respiration rate of 40 L/kg/hr (hard labor) as
well as a conservative 100% absorption in the lungs, a 8 hr.
working day at the recommended TWA would give rise to an
absorption of 67.2 mg. Since in this case, a safety factor of 10
would seem appropriate, this will give an "ADI" identical with
the proposed AIC value above.

B. Di-n-butylphthalate (DBP)

Phthalates seem to be readily absorbed in the gastrointestinal
tract with concomitant formation of the corresponding monoesters
(Albro, P.W. and More, B., J. Chromatogr. 94(1974)209-218).
Excretion occurs mainly via urine as monoester or its oxidation
products. Four different oxidation products of monobutyl
phthalate have been identified from DBP and no significant
metabolic species differences were noticed in the rat, the
hamster and the guinea pig (Tanaka, A. et al, Toxicol.
9(1978)109-123). However, based on studies on other phthalates
the metabolism in primates would be expected to differ.

The acute and the chronic toxicity of DBP appear to be very low.
No adverse effects were noted in a French study covering 3 to 5
generations of rats fed diets containing 100-500 mg/kg
(LeBreton, M. cited in LeFaux, R. The Practical Toxicology of
Plastics, CRC Inc., Cleveland, Ohio, 1968, pp. 138-139).
Although this study would seem inadequate by modern standards in
several respects, it nevertheless indicates a low level of
chronic toxicity and the results seem to be substantiated by a
1-year study in rats where doses of 110-350 mg/kg did not affect
growth and survival (Smith, C.C. Ind. Hyg. Occup. Med.

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8(1953)310-318). Enlargement of the liver and spleen could be observed in rats administered 1 ml/kg/day or 5 ml/kg/day DBP for 3 weeks (Yamada, A. Shokuhin Eiseigaku Zasshi 15(1974)147-152 cited in CA 083/054100Q). In a another study kidney and liver damages were inducted in mice receiving 0.5 - 5.0 g/kg/day orally for 1-3 months (Ota, H. et al, Nippon Eiseigaku Zasshi 29(1974)519-524 cited in CA 083/0541815).

In a more recent investigation where pregnant ICR-JCL mice were administered a diet containing 0.0, 0.05, 0.1, 0.2, 0.4 or 1.0% DBP, decreased maternal weight gain and an increased resorption rate at the 1% level (2100 mg/kg/day) was seen (Shiota, K. et al, Environm. Res. 22(1979)245-253).

No evidence of carcinogenic effects have been reported. On the other hand no fully adequate long-term study seem to have been performed.

As with DEHP, high doses (2g/kg for 4 days) of DBP causes testicular toxicity (Cater, B.R. et al Toxicol. Appl. Pharmacol. 41(1977)609-618). Although an increase in sister chromatid exchanges has been reported in Chinese hamster cells exposed to DBP (Abe, S. and Sasaki, M., J. Natl. Cancer Inst. 58(1977)1635-1641) investigations in other short-term test systems have been negative (Shahin, M.M. and von Borstel, R.C., Mut. Res. 48(1977)173-180). Teratogenic effects have been observed upon i.p. injection of DBP in high (more than 0.3 ml/kg) doses to Sprague Dawley rats (Singh, A.R. et al, J. Pharmacol. Sci. 61(1972)51-55).

Risk Assessment: The current TWA adopted by ACGIH for 1985-86 is 5 mg/m^3. In the Draft Superfund Public Health Evaluation Manual of December 18, 1985 a minimum effective dose (MED) for selection of indicator chemicals of 420 mg/kg but no AIS or AIC values are given. If the lower dose levels of 100-110 mg/kg/day in the study of LeBreton and Smith cited above are used as the NOAEL and a safety factor of 100 employed, a "tentative ADI" of 1 mg/kg/day is obtained which would not seem unreasonable considering the general toxicity profile of similar compounds.

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However, in view of the incompleteness of the long term toxicity
studies and a suspicion of some teratogenic potential, it may be
prudent to use a larger safety factor. In a criteria document
published by the Environmental Health Directorate (Health and
Welfare Canada, Phthalic Acid Esters, 80-EHD-62, p. 109) a safety
factor of 500 was used bringing down the provisional ADI to 0.22
mg/kg/day.

C. 1,1-Dichloroethane

In experimental-animals this substance induces signs of CNS
depression at high doses (has been used as an anaesthetic), and
damage to liver and kidney has been demonstrated in experimental
animals as well as in man (Parker, J.C., et al, Am. Ind. Hyg.
Assoc. J. 40(1979)A46-A60).

In a long term study in rodents (NCI) 1,1-dichloroethane was
administered by gavage for 78 weeks, but the results were
considered inconclusive with respect to carcinogenicity due to
poor survival. However, the data do seem to permit a reasonable
estimate of ADIs based on non-carcinogenic effects.

In the final draft report on exposure and risk assessment for
this compound, by Arthur D. Little, it is suggested that due to
the compounds structural similarity to 1,2-dichloroethane and
because of a marginal increases in some tumor types in this
study, 1,1-dichloroethane should be considered as a suspect
carcinogen until proven otherwise (EPA 440/4-8/009).

For the following reason this conclusion does not seem justified:

1.   Data from the study have been judged inadequate for any
     inferences as to possible carcinogenic action.

2.   No adequate evidence from short-term tests is
     available.

3.   The structure-activity comparison with
     1,2-dichloroethane is not warranted. The carcinogenic
     activity of 1,2-dichloroethane seem to be uniquely

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> linked to alkylation of endogenous substances like
> glutathione giving rise to half-mustards, R-S-CH$_2$CH$_2$Cl,
> which in turn may act as potent genotoxic alkylating
> agents. This specific reaction pathway would not apply
> the 1,1-isomer. After metabolic activation,
> 1,2-dichloroethane is clearly mutagenic in a number of
> systems in contrast to many other chloroalkanes and
> chlorolkenes.

Risk Assessment: ACGIH has adopted a TWA value of 200 ppm (810
mg/m^3). The Superfund Public Health Evaluation Manual of
December 18, 1985 lists a subchronic acceptable daily intake
(AIS) of 1.2 mg/kg/day and a chronic acceptable daily intake
(AIC) of 0.12 mg/kg/day. Corresponding values by the inhalation
route are listed as 1.38 mg/kg/day and 0.14 mg/kg/day
respectively.

D. Cis-1,3-dichloropropene

In view of the low stability of 1,3-dichloropropene, the
detection of this compound in appreciable quantities off the
Tyson dump site is surprising. Thus, pure 1,3-dichloropropene
without added stabilizer was not considered sufficiently stable
to be used in a carcinogenesis study (Schwetz, B. of NTP, in
Toxicology and Carcinogenesis Studies of Telone II, NTP Techn.
Rept. Ser. No. 269, May 1985, p.14). 1,3-Dichloropropene has
been reported to have a half-life in soil of about 10 days
(Laskowski, D. et al, Terrestrial environment, Environ. Risk.
Analy. chem. 25(1982)198-240). The substance is hydrolyzed in
wet soil to 3-chloroallyl alcohol (Castro, C. and Belser, N.J.
Agric. Food Chem. 14(1966)69-70). In an area in California where
1,3-Dichloropropene had been used in agriculture as a fumigant
for years, 54 wells were analyzed for residual amounts of the
compound without finding measurable amounts at a minimum
detectable level of 0.1 ppb (Cf. NTP Techn. Rept. Ser. No. 269,
May 1985, p.16-17).

Due to its instability, 1,3-dichloropropene used as fumigant in
agriculture as Telone II (Dow Chemical Co.) contains 1%
epichlorohydrin as a stabilizer.

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After oral administration, the compound is rapidly excreted by a metabolic pathway that involves conjugation with glutathione, further transformation to a mercapturic acid, and excretion mainly as N-acetyl-S-[(cis)-3-chloroprop-2-enyl]-cystein.

The acute toxicity of Telone II is moderate with an oral LD50 of about 500 mg/kg in the female rat. Liver and kidneys are the primary targets of acute toxic effects. Subchronic inhalation experiments in rats at 11 or 50 ppm for 7hrs/day, 5 days/week during 1 month likewise caused liver and kidney damage.

Positive results have been obtained in short-term testing, but it seems that the presence of impurities like epichlorohydrin may have been responsible for these effects.

1,3-Dichloropropene has been studied in a long term carcinogenicity study (gavage in corn-oil) under the NTP program in mice (B6C3F1) and rats (F344/N). The study in male mice was considered inadequate because of a markedly reduced survival in controls. There was also a failure to fully randomize the animals in the mouse studies. A small increase in the incidence of squamous cell papillomas and carcinomas in the forestomach were noted in rats which was statistically significant in males. There was also a small, but statistically significant increase in transitional cell carcinomas of the urinary bladder, of papillomas and carcinomas of the forestomach, as well as an increase in adenomas of the lund in female mice. NTP has judged that the study provides clear evidence of carcinogenicity for male rats and female mice, and some evidence of carcinogenicity in female rats. According to the principles laid down by IARC, the evidence is not sufficient to support an assumption of carcinogenicity for 1,3-dichloropropene. As mentioned under A.5.3, OSTP has clearly spelled out that a bioassay in which the test substance is not free from carcinogenic contaminants cannot be considered as valid for assessment of the compound itself. The role of epichlorohydrin (which causes tumors of the forestomach) as initiator in induction of tumors in presence of a chlorinated hydrocarbon promoter has been discussed under Section A.5.7. The irrelevance of forestomach tumors in the nonglandular

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forestomach of rodents to the corresponding fundus portion of man
was commented upon under Benzo(a) pyrene, Section A.6. The NTP
summary states that "the 1.0% epichlorohydrin, a direct-acting
mutagen and carcinogen added as a stabilizer, may have influenced
the development of forestomach lesions."

As to the carcinogenicity of 1,3-dichloropropene, the increase of
adenomas and of carcinomas of the lung of female mice must be
considered as inadequate evidence in view of the following facts:

- A finding of carcinogenicity in one sex in one strain
  of a species of experimental anaimals can never be
  accepted as sufficient evidence.

- Here, as well, the 1,3-dichloropropene could exert a
  promoting effect on cells initiated by epichlorohydrin
  or by other endogenous initiators.

Rish Assessment: Until more convincing data are available,
1,3-dichloropropene should be treated as a noncarcinogen. In
rats no major non-neoplastic findings of toxicity were observed
at any two dose levels used (25 and 50 mg/kg). However, there
was a marginal depression of mean body weight (5 - 9%) in the
high dose in males. The data in mice were difficult to interpret
due to the failure of randomization and the high rate of
myocarditis in the male controls. If 25 mg/kg is taken as the
NOEL, an AIC of 0.25 mg/kg/day is obtained.

E. Chlorobenzenes

Although some of the chlorinated benzenes undergo microbial
degradation, they are lipophilic compounds which to a smaller
or greater extent bioaccumulate in biota. Special attention
therefore should be paid to their toxicological properties. The
toxicology of chlorobenzenes has recently been well reviewed by
EPA in an extensive health assessment document
(EPA/600/8-84/015F).

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Monochlorobenzene: Although quantitative data seem to be
lacking, available evidence indicate that monochlorobenzene is
readily absorbed by the oral route or by inhalation. It is
rapidly distributed to many tissues (with a significant
deposition in fatty tissues) and metabolized into a variety of
products, presumably via epoxides which are converted to
chlorophenols (mainly monophenols) and to conjugation products of
phenols with glutathione, glucuronic acid or sulphate. By the
action of epoxide hydrase, dihydrodiols can also be formed which
in turn may give chlorocatecols. Chlorobenzene-induced necrosis
of the liver may be prevented by inhibiting the epoxide hydrase
which seem to indicate the formation of reactive intermediates
along the dihydrodiol pathway (Oesch, F. et al, Chem. Biol.
Interact. 6(1973)189-202). The metabolic saturation
demonstrated at relatively low exposure levels is significant
when assessing the toxicity of this compound. The principal
metabolites in humans are the same as those seen in animals.

The acute toxicity of monochlorobenzene is relatively low, where
necrosis of the liver and proximal tubuli of kidneys observed in
experimental animals after exposure to high doses may be
associated with the covalent binding of metabolites by proteins
in these organs.

The signs of chronic toxicity in animals include urinary
prophyria, leisions of liver, kidney, spleen, thymus as well as
bone marrow injury. Repeated exposure by inhalation or by the
oral route has also been shown to cause tissue atrophy of the
seminiferous tubules and decreased spermatogenesis in dogs and
rats at high doses.

Although no chronic inhalation studies seem to have been carried
out, results from a number of subchronic inhalation studies have
been published. In one study in rats and rabbits involving
exposure 7hrs/day, 5day/week, for 24 weeks, 77 ppm appeared to
represent the marginal toxic concentration for daily inhalation.
However, neurotoxic effects have been described by Soviet
investigators at much lower chronic exposure levels (0.2 ppm for
60 days of continuous exposure; Tarkhova, L.P., Gig. Sanit.

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30(1965)327-333), which may account for the lower TLVs
adopted in some of the Socialist countries.

In a long term NTP oral study in rats and mice no significant
increase incidence of tumors was found. Results from short-term
tests have been mainly negative.

Risk Assessment: The current OSHA TWA value for
monochlorobenzene as well as the recommended ACGIH TWA is 75 ppm
(350 mg/m^3).

The Draft Superfund Public Health Evaluation Manual of December
18, 1985 an AIC of 0.027 mg/kg/day is cited, i.e. of an order of
magnitude lower than the AIC for a much more toxic compound such
as carbon disulfide. The value for monochlorobenzene does not
seem to be based on the relevant NTP studies and should be
rejected.

In the NTP long-term studies cited above the highest dose tested
in rats, 120 mg/kg/day, induced an increase in neoplastic nodules
in males, which could be taken as the LOAEL. At 60 mg/kg/day no
signs of adverse toxic reactions could be detected in either mice
or rats and this level seems to adequately represent the NOAEL in
these studies, giving an ADI of 0.6 mg/kg/day for
monochlorobenzene.

1,2,4-Trichlorobenzene: Limited data on pharmacokinetics and
metabolism are available for the trichlorobenzenes. This isomer
seems to be readily absorbed from the intestinal tract, by
inhalation and to some extent also upon skin contact with an
expected preferential distribution to fatty tissues. Similarly
to monochlorobenzene, metabolic conversion to trichlorophenols
(major metabolites), catechols and conjugates of glucuronic,
mercapturic, and sulphuric acids occurs. In the monkey
1,2,4-trichlorobenzene is metabolized to an isomeric pair of
3,4,6-trichloro-3,5-cyclohexadiene-1,2-diol glucuronides (48-61%).
Thus, important metabolic species differences may exist for this
substance.

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The acute toxicity of 1,2,4-trichlorobenzene is relatively low.
In subcronic and chronic toxicity studies, oral administration of
toxic doses has revealed kidney and liver toxicity (necrosis,
fatty infiltration, hepatomegaly) accompanied by porphyria. No
adequate long-term study for the evaluation of potential
carcinogenicity seem to have been performed.

Risk Assessment: There are no US workplace standards for the
trichlorobenzenes, but the ACGIH has recommended a ceiling of 5
ppm (40 mg^3) and NIOSH has designated these substances as
Group III pesticides. No AIS or AIC data for
1,2,4-trichlorobenzene has been listed in the Draft Superfund
Public Health Evaluation Manual of December 18, 1985. However,
the subchronic toxicity of 1,2,4-trichlorobenzene has been
investigated in several species, and from the data available a
subchronic acceptable intake (AIS) may be derived. In an
inhalation study in Sprague Dawley rats at 0, 22.3, or 74.2 mg/m^3
(10 ppm) for 6 hrs/day, 5 days/week for 3 months, 22.3 mg/m^3 did
not increase porphyrin excretion - evidently the most sensitive
indicator of toxicity in rats - and this level was considered as
a NOAEL (Watanabe, P.G. et al, Toxicol. Appl. Pharmacol.
45(1978)322-333). For monkeys and rabbits a NOAEL of 742 mg/m^3
was established in an inhalation study with a 26 weeks duration
(7 hrs/day, 5 days/week; Coate, W.B. et al, Arch. Environm. Hlth.
(32(1977)249-255). EPA has reviewed the data from oral
administration in monkeys for 30 days where no toxic effects were
observed at 25 mg/kg, while doses of 90 mg/kg or more were toxic.
174 mg/kg was lethal within 20-30 days. However, due to
inadequate reporting and other circumstances, according to the
Agency no valid NOEL can be derived from these data (cf. EPA,
Health Assessment Document for Chlorinated Benzenes,
EPA/600/8-84/015F, p. 9-21).

Based on the study of Watanabe et al in the rat and assuming 50%
absorption in the lungs as well as a respiratory rate of
30 L/kg/hr in this species, an inhalation AIS of 0.08 mg/kg/day
can be derived. In view of the results from the monkey study,
which gave a considerably higher NOAEL, and taking into account
the great metabolic species difference where the primate accounts
for the excretion of an appreciable fraction of a species

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specific metabolite, this AIS value could be regarded as too low.
However, the value is proposed as a conservative estimate which
obviously also can be used as upper limit for the oral AIS.

F.   Phenol

Phenol is a relatively toxic chemical that may be absorbed by
ingestion, dermal contact or by inhalation. The substance is
mainly excreted as pheylsulfuric and phenlglucuronic acids,
oxidized to catechols and quinones or to carbon dioxide.
Elimination of free phenol also occurs. It has been suggested
that signs of toxicity only appear after absorption of doses
which may produce metabolic overloading (NIOSH, Criteria for a
Recommended Standard - Occupational Exposure to Phenol, July
1976, p. 62).

Phenol can cause local as well as systemic effects. It is
corrosive to skin and when in contact with the eye it can cause
severe damage or even blindness. Concentrated solutions of
phenol have cause death after skin contacts as brief as 5-20
minutes.

Symptoms of acute intoxication include weakness, sweating,
headache, giddiness, etc., which can be followed by cyanosis,
convulsions, coma and death (usually from respiratory failure).
If death does not occur, kidney damage may be elicited. Dark
colored urine is often a characteristic sign of poisoning.

Chronic exposure has been reported to cause loss of appetite,
anorexia, dizziness, diarrhea, dark urine, discoloration of the
skin, mental disturbances, and signs of liver toxicity
(Merliss, R.R., J. Occup. Med. 14(1972)55-56).

In a three generation study in rats 5000 ppm in drinking water
was said to produce no adverse effects, whereas stunted growth
was evident in the young of a group exposed to 7000 ppm in water
over 2 generations. The offspring of rats at 10000 ppm died at
birth (Heller, V.G. and Pursell, L.J. Pharmacol. Exptl. Therap.
63(1938)99-107). No toxic effects could be found in monkeys,
rats and mice exposed to 5 ppm (19 mg/m^3) in the inhaled air

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8 hrs/day, 5 days/week for 90 days (Sandage, C., ASD Technical
Report 61-519, Kansas City, Mo. Midwest Research Inst., 1961,
pp. 1-29, cited in NIOSH, Criteria for a Recommended Standard -
Occupation Exposure to Phenol, July 1976, pp. 55-60). 26-52 ppm
(100-200 mg/m^3) clearly represents a toxic level to guinea pigs
since 29 such exposures killed 5 out of 12 animals. Rabbits and
rats were found to be much less sensitive (Deichamnn, W.B.,
et al, Am J. Clin. Pathol. 14(1944)273-277. Phenol did not
induce carcinogenic effects when administered in drinking water
to rats and mice in a long term NTP study. Phenol possesses
warning properties by odor and taste far below the concentrations
at which toxic effects occur (odor threshold 0.02-0.19 mg/m^3;
NIOSH, Criteria for a Recommended Standard - Occupational
Exposure to Phenol, July 1976, p. 68).

Risk Assessment: NIOSH and the ACGIH has recommended a TWA of
5 ppm (19 mg/m^3. In the draft Superfund Public Health Evaluation
Manual of December 18, 1985 an AIS as well as an AIC of 0.1
mg/kg/day has been given for phenol.

G. Selenium

Since the selenium found at the site was associated with soil,
the toxicology of inorganic selenium compounds other than
hydrogen selenide will be considered relevant.

Selenium is an essential element where the margin of safety
between "desirable" exposures and toxic levels is rather narrow.
Deficiency in selenium will cause disturbances in the cellular
protective defense mechanism against oxidative stress. The role
of selenium as a constituent in glutathione peroxidase has
received particular attention. Pathological signs of deficiency
in mammals include inter alia liver necrosis and cardiomyopathy.
The element has also been implicated as an anticarcinogen in
several epidemiological and experimental studies.

The bioavailability of selenium is dependent on its chemical
form; elemental selenium and selenides of heavy metals are very
insoluble. Monogastric animals have a high intestinal absorption
(more than 90% absorption of selenite in man) and is rapidly

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distributed to various organs. Some selenium compounds, like
selenium oxychloride, are strong vesicants causing destruction of
the skin and considerable dermal uptake. However, such compounds
are not likely to be of interest in this context. Selenium may
be biotransformed by methylation or incorporation in amino acids
(e.g. Se-methionine). Dimethyl selenium is an intermediate in
the formation of urinary trimethyl selenium which is exhaled
(garlic breath) in acute intoxication. Excretion is composed of
a rapid phase of excretion of 15-40% of the absorbed dose during
the first week, followed by a slow elimination with an
approximate half-life of 100 days. Cumulative effects may, thus,
be expected upon metabolic overloading.

Acute selenium poisoning in man is primarily characterized by
toxic effects on the central nervous system which sometimes
include convulsions. Inhalation of larger amounts of selenium
dioxide may cause pulmonary oedema.

Chronic intoxication gives rise to vague subjective symptoms like
gastrointestinal stress, nervousness, lassitude as well as
partial loss of hair and nails and discoloration of teeth.
Evidence of chronic selenium intoxication by oral administration
in man has been found in seleniferous areas. From chronic
occupational over exposure, liver and spleen damage as well as
anemia have been described. In life stock ("alkali" disease)
excess selenium in feed in the range of 25 ppm causes anorexia,
loss of hair, atrophy of hooves, lameness, sterility, fatty
necrosis of the liver, and anemia. Selenium has also caused
embryotoxic as well as teratogenic effects in experimental
animals.

In high doses selenium sulphides induce an increase in
hepatocellular carcinomas and adenomas in male and female rats
and female mice as well as pulmonary tumors in female mice. NTP
has judged the animal evidence of carcinogenicity as sufficient.
However, in this case the EPA has evidently taken the position
that selenium may only pose a cancer risk at high doses or
that the rodent model is not valid here. The compound is not
listed as a carcinogen in the Superfund Public Health Evaluation

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Manual of December 18, 1985. Selenium is consequently permitted
as a feed additive in the US.

Risk Assessment: The current drinking water standard (MCL)
according to the Safe Drinking Water Act is set at 0.01 mg/L.
Considering available toxicological information as well as
current work place standards, this value seems to be unduly
conservative.   Thus, the Federal Standard for occupational
exposure is given as 0.2 mg/m^3 which implies that exposure to
inorganic selenium at that TLV may give an absorption of about
0.06 mg/kg/day (assuming 50% absorption in the lungs). In line
with this conclusion, the recently proposed recommended MCL has
been raised to 0.045 mg/L, giving a maximum daily intake of 0.09
mg, and in the Superfund Public Health Evaluation Manual of
December 18, 1985 oral AIS and AIC values are given as 0.003
mg/kg/day and the inhalation AIC value as 0.001 mg/kg/day.

H.   Zinc

Zinc is an essential element, and in man the average daily intake
of zinc is about 10-15 mg/day.

By the oral route or by inhalation the acute as well as chronic
toxicity of inorganic zinc is extremely low.   Zinc salts of
strong mineral acids are astringents and solid zinc chloride is
corrosive to skin and mucous membranes. Respirable particles of
zinc oxide (or hot zinc fumes in presence of oxygen) may cause
metal fume fever. However, these are effects of little interest
in the current context.

Drinking acidic beverages made in galvanized containers has
caused signs of acute poisoning involving vomiting and diarrhea.
Only by the parenteral routes do zinc salts have an appreciable
toxicity.

In chronic animal experiments high concentrations in food or
drinking water are tolerated. Symptoms of toxicity after
subchronic or chronic exposure at dietary levels above 0.1-0.5%
include anemia as well as impaired growth and adverse effects on
reproduction.

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Testicular tumors have been induced by direct intratesticular
injection in rats, a finding of questionable significance for
risk assessment in man; and zinc salts administered by other
routes have not produced carcinogenic effects (Furst, A.,
Environm. Hlth. Perspect. 40(1981)83-91). The antagonistic
effect of zinc in relieving certain effects of intoxication
caused by agents like cadmium is well documented.

Risk Assessment: The present TWA for zinc oxide fumes is 5 mg/m^3.
In the Superfund Public Health Evaluation Manual of December 18,
1985 the listed oral AIS and AIC values for zinc and zinc
compounds are 0.21 mg/kg/day, whereas the corresponding value for
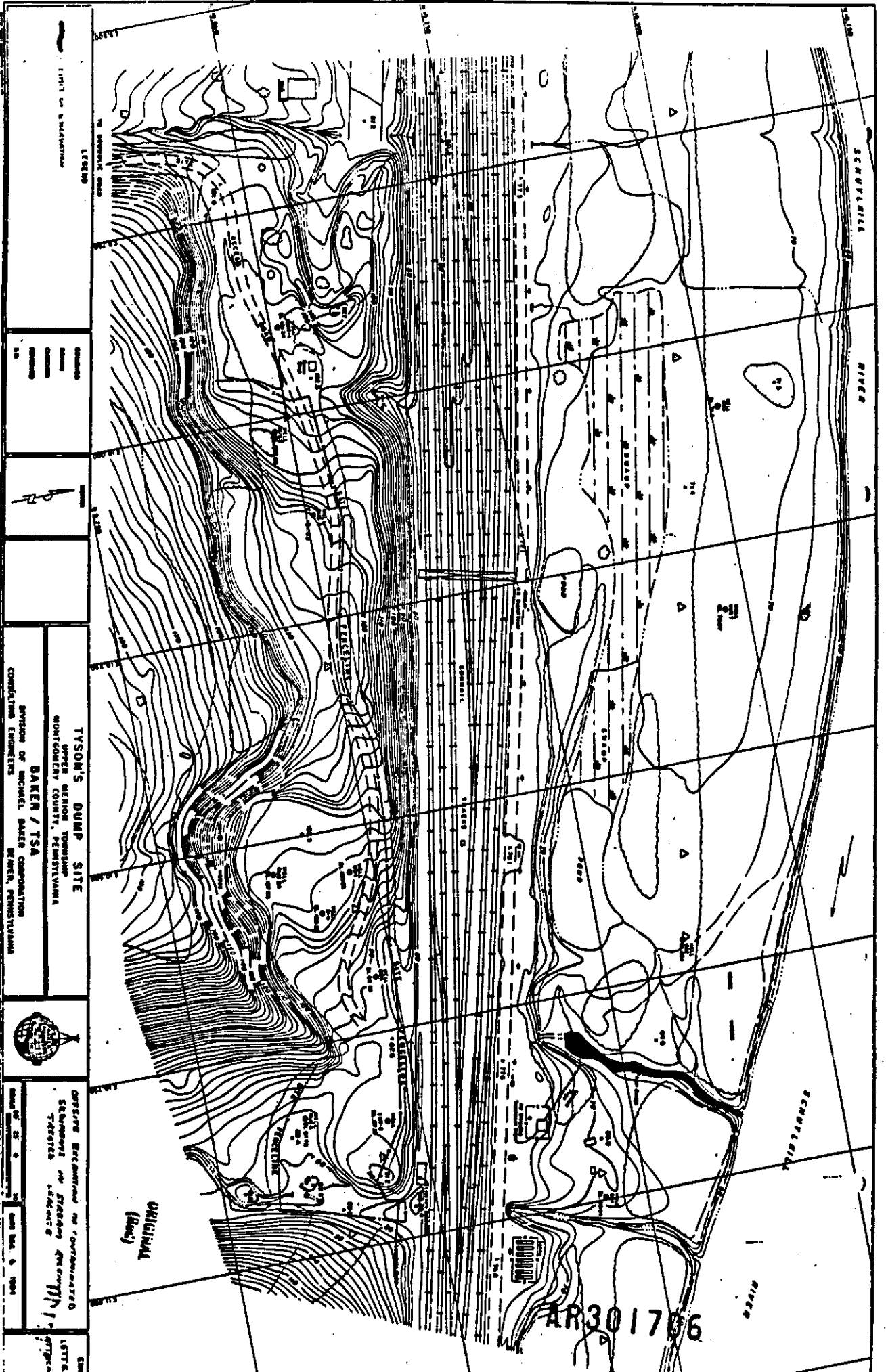the inhalation route is 0.1 mg/kg/day.

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B.3 SUMMARY

In the table below a summary of suggested acceptable subchronic (AIS) and chronic intakes (AIC) for the selected non-carcinogenic indicator substances are listed:

Compound	Acceptable daily intake mg/kg/day	Comments
Carbon disulfide	0.1	Oral AIC (EPA)
Di-n-butyl-phthalate	1.0	Oral AIS (proposed here); No EPA standards
1,1-Dichloro-ethane	1.2 0.12	Oral AIS (EPA) Oral AIC (EPA)
cis-1,3-Dichloro-propene	0.25	Oral AIC (proposed here); No EPA standards
Monochloro-benzene	0.6 0.027	Oral AIC (proposed here); Oral AIC (EPA)
1,2,4-Trichloro-benzene	0.08	Oral AIS (proposed here); No EPA standards
Phenol	0.1	Oral AIS and AIC (EPA)
Selenium (inorganic)	0.003	Oral AIS and AIC (EPA)
Zinc	0.21	Oral AIS and AIC (EPA)

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